HANDBOOK OF OBSTETRICS AND GYNAECOLOGY

by

Thomas J. Borody M.B. B.S. B.Sc. (Med.)

Roderick D. Peek M.B. B.S. B.Sc. (Med.)

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With Illustrations
by
Susan Casey

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FOREWORD

In 1972 the School of Obstetrics and Gynaecology at the University of New South Wales introduced a new curriculum and assessment scheme based on modern educational principles.
The basic concept was to develop many workable Units of Instruction containing relevant material which could be learnt in about 10–20 hours of structured student activity. Each Unit of Instruction had an overall aim stating the fundamental concept to be learnt, the reason for learning the material and the level of competence to be achieved. The aim is called the General Instructional Objective for the Unit of Instruction.

A series of Learning Strategies was then developed for each Unit of Instruction, stating clearly where the student was most likely to learn the relevant material to achieve the competence required for the General Instructional Objective.

Finally, a sample of the behaviours a student should exhibit, to show he has understood, and can achieve, the General Instructional Objective, are listed. The samples of the Specific Behaviours which the student must exhibit at the completion of the course contain representative material from the three domains of knowing (cognitive), doing (psychomotor) and careing (affective). When assessing whether the student has achieved the required standard of competence in the course, only the Specific Behaviours are examined, and because a large number of the behaviours are not examinable by conventional means a new type of assessment was devised based on student/patient interaction and problem-solving devices (Patient Management Problems).

When the students first entered the course, they were given a copy of the new curriculum and a comprehensive program was drawn up to allow greater student/patient contact. A series of introductory lectures was given at the beginning of the term followed by multiple small group tutorials based on provocative cases. Notes and reference books were provided but the students felt that a large amount of material was not adequately explained regarding its relevance, and where differing opinions occurred, confusion among students became a source of frustration.

During the first term of 1974, three students (Tom Borody, Cliff Rosendahl and Rod Peek) suggested that they write a handbook to explain, in reasonable detail, the various points which required clarification. They were eventually persuaded to write this handbook covering all the Units of Instruction so that students coming late to the term could easily read and organise their thoughts on any of the Units of Instruction. On completion of the text the illustrations were kindly prepared by Susan Casey, another medical student.

This handbook is not intended to replace the notes or the reference books but is to act as a means of quickly identifying the interrelationship of the content material in each Unit of Instruction.

Although it was prepared with the course in obstetrics and gynaecology at the University of New South Wales in mind, it may be of value to other students.

Each chapter begins with the curriculum plan clearly set out for a specific Unit of Instruction and the handbook summarises the basic cognitive knowledge required in that field of instruction. However, as previously stated, this handbook does not replace the notes or reference books, nor does it replace the practical application, of the skills and knowledge to be learnt, to real-life situations. I hope students will find this handbook useful.

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CHAPTER 1

HISTORY TAKING AND PHYSICAL EXAMINATION

General Instructional Objective

Develops competence in history taking and physical examination so that normality and abnormality can be recognised in the obstetrical or gynaecological patient.

Specific Behaviours

1. Demonstrates an ability to elicit all the relevant history.
2. Demonstrates an ability to competently examine a patient.
3. Demonstrates empathy in taking a history and conducting a physical examination.
4. Describes observations made during physical examination of a patient.

History Taking and Physical Examination

Obstetric History

Name and marital status

Age

Parity (e.g. Para. 1+1 means one delivery of more than 20 weeks gestation plus one delivery of an abortus of less than 20 weeks gestation).

Last menstrual period (L.M.P.)	Record first day of last menstrual period, and ask about its duration, volume of flow and pain. Did it appear normal, or was it merely spotting. If the latter is the case then the second last menstrual period should also be recorded in case last menstrual period was really an implantation bleed.
Cycle	(e.g. 5/28-38) Ask whether patient was on the "Pill" shortly before falling pregnant and, if so, whether regular periods had been reestablished since cessation of "Pill".
Expected date of delivery (E.D.D.)	Method of calculation in a 28 day cycle: e.g. L.M.P. 3.8.73 add 10 days and 9 months E.D.D. 13.5.74 If cycle is longer than 28 days, the E.D.D. is brought forward by the extra number of days in the period. The converse is done if the cycle is shorter.
Number of weeks amenorrhoea	As calculated from L.M.P.
Nationality	Important in e.g. Thalassaemia; T.B.
Anticipated overseas trip	Patient will need to be advised re contra-indicated immunisations.

Past History

Obstetrical:

Pregnancy No.	Year	Place	Maturity Reached	Labour Length	Delivery Nature	Complications	Sex	Wt.

Gynaecological:

Menstrual history	Age at menarche. Menstruation since then.
Contraceptive	History. Details of all contraceptives used. Was any contraceptive used when the present pregnancy began?

Medical:	Any heart disease, rheumatic fever, hypertension. Any T.B., asthma, bronchitis. Any Urinary Tract Infection or Pyelonephritis.
Surgical:	Any operations or accidents. Any D & C's (often not regarded by the patient to be an operation). Any transfusions. *N.B.* With respect to D & C's or other operations of significance, record the date, place and name of the H.M.O. involved.
Family Health	Any serious illnesses in close relatives. Any T.B., diabetes in the family. Any history of twins.
Social	If patient is single, ask what her plans are for the infant. Do you think the patient needs to see a Social Worker. Is she a member of a Medical Benefits Fund?

History of Present Pregnancy

Ask about the common symptoms of pregnancy:

Nausea and vomiting.
Breast swelling and tenderness.
Frequency of micturition – is there any scalding.
Fainting.
Heart burn, etc.

Ask about foetal movements:

These are first felt at 16–18/52, amenorrhoea in multigravidas and at 18–20/52 amenorrhoea in primigravidas.
The exact day that they are first felt should be recorded if possible. Movements are first felt as faint sensations of "bubbles" or "butterflies in the abdomen."

Abnormal symptoms and influences:

Ask about pain, P.V. bleeding, and abnormal discharge.

Ask about abnormal urinary tract and bladder symptoms, e.g. urine retention.
Ask about any infection at all, e.g. respiratory tract infection.
During the 2nd and 3rd trimesters, ask about headache, visual disturbance, and finger oedema (does her wedding ring feel tighter).
Ask about any drugs or medications.

Is she taking iron and folate tablets and, if so, does she have enough to last until the next visit.

System Review

Ask about:	Medications or drugs at present.
	Allergies.
	Smoking.
	Alcohol intake.
	Weight; normal and any change.
	Last Pap. smear.

Now investigate systems in detail as indicated by previous findings.

Obstetrical Examination

General impression	Note anything obvious; height; weight.
Hands and nails	
Radial pulse	Blood measure
Feet	Swelling; varicosities
Teeth and oral hygiene	
Thyroid	Any enlargement
Thorax	Inspect and auscultate the heart and lung fields.
Breasts	Inspect – Any darkening of the areola. Any enlargement of Montgomery's tubercles. Any increase in venous engorgement. Palpate – Any fluid expressable from the nipples. Any lumps.

Abdomen	Inspect striae, scars, anything obvious.
	Palpate liver, spleen, kidneys, uterus. If the uterus is palpable, then estimate as much of the following as possible: (none of these observations will be relevant until close to term)
Fundal height	Just above the symph. pubis (12/52); Midway from symph. to umbilicus (16/52); Just above umbilicus (24/52). Divide area from umbilicus to xiphisternum into thirds. Fundal heights at the upper level of each third are consistent with 28, 32 and 36 weeks amenorrhoea respectively. After 36/52 the fundus may descend to a variable degree depending on whether the head engages so that at 38/52, for example, the fundal height may also be consistent with 34/52. State your overall impression as to uterine size.
Foetal lie	Longitudinal, oblique or transverse (relationship of long axis of foetus to long axis of uterus).
Presentation	Cephalic, breech or otherwise.
Position	Relationship of denominator of the presenting foetal part to the mother's pelvis. As usual presenting part is the head, then the occiput is the usual denominator (face – mentum, breech – sacrum). The denominators used to describe positions are: · Occipital – in a vertex presentation. · Mental – in a face presentation. · Sacral – in a breech presentation.
Attitude	Normally universal flexion.
Engagement	Engagement is said to have occurred when the widest diameter of the presenting part has passed through the pelvic brim. This may be determined by palpation.

If the presenting part is not engaged, determine whether it can be engaged by *gentle* abdominal pressure.

Foetal heart — Is it audible and, if so, where is the point of maximum intensity.

Notes on determining the foetal position if in vertex presentation

Side of occiput — Left 40%
Right 35%
(The *right* occipital position is used below to illustrate OA, OL, and OP positions).

ROA — Back on right of midline.
Anterior shoulder within 5 cm. of either side of the midline. This corresponds with the point of maximal intensity of foetal heart sound.

ROL — Back on right side – further towards the flank.
Anterior shoulder more than 5 cm. to the right of the midline – corresponds with the point of maximal foetal heart intensity.

ROP — Back well out into flank on right side, shoulder in the flank.
Foetal heart sound maximal in right flank or over midline.
Hollowing of the abdomen just below umbilicus.
Back not palpable.

Pelvic Examination — Always done at the first visit and later if indicated.

Speculum Examination — First inspect the perineum for ulcers, tumours, scars or evidence of scratching.
Using the left hand (if right handed), separaet the labia majora and inspect the vestibule and urethra.

Holding a suitably sized speculum (which you have lubricated) in your gloved right hand and holding the labia separated with the thumb and index finger of the left hand, gently insert the speculum all the way (as long as this does not cause any severe discomfort). The speculum may then need to be withdrawn a little for the cervix to be visualized.

Observe the following features of the cervix:

1. Site : Midline or otherwise.
2. Size :
3. Contour : Irregularities, tumours.
4. Colour : Is it darkened (bluish) as in pregnancy.
   Is there any ectopic columnar epithelium (the cervix is normally covered with flat pink squamous epithelium)?
5. Os : Nulliparous (round) or parous (transverse).
6. Discharge: If present, is it coming from the os.

Before closing the speculum draw it back clear of the cervix.

When it is closed draw it gently from the vagina and place it in the bowl provided.

Digital Examination — Using the gloved right hand, insert first one finger and then two, gently into the vagina. Palpate the vaginal walls.

Now place the left hand just above the pubis and bimanually palpate the cervix, uterus and lateral fornices.

Pelvic adequacy may be assessed.
(Often not done till 36/52).
Method: Reach diagonally upwards and backwards towards the sacral promontory (assessing the diagonal conjugate diameter). If the promontory cannot be reached the diameter is usually adequate.
Palpate as much of the brim as possible, working from the front backwards.
Next assess the cavity by palpating the sacrum.

The lower two or three segments may be palpable.
Is the sacrum flat or concave?
Palpate the ischial spines and note whether they are prominent.
To assess the outlet first estimate the subpubic angle. It should normally be at least 85°.
Estimate the bituberous diameter. It should be at least 10.5 c.m. If it is reduced or the sub-pelvic angle is narrow, estimate the post saggital diameter. This is measured from the midpoint of the bituberous diameter to the tip of the coccyx, and should be at least 7.5 cm.

Withdraw the examining fingers, and examine them for blood or discharge which has adhered.

Record:

Vagina — Normal, hypertrophic or atrophic epithelium. Any cystocele, rectocele, urethrocele, etc.

Cervix
- Site :
- Contour : Irregularities, tumours, etc.
- Consistency : Is it softened as in pregnancy?
- Mobility :
- Tenderness :
- Os : Parous or nulliparous.

Uterus
- Site : Midline or otherwise.
- Direction :
- Size : "Lemon" 7/52 amenorrhoea. "Orange" 10/52 amenorrhoea. "Grapefruit" 12/52 amenorrhoea.
- Shape : Contour, irregularity, fibroids.
- Consistency : Firm, hard or soft.
- Mobility :
- Tenderness :

Fornices and Adnexae — If there is nothing obvious to be found then there are probably no adnormalities. Palpation of the ovaries is difficult unless the patient is under anaesthesia.

Gynaecological History

Name and marital status

Age

Parity

Last menstrual Period and second last menstrual period

Menstrual cycle,

Presenting Symptoms — Try to ascertain what is worrying the patient *most.*

History of Present Condition — Pursue this in a logical sequence, but finish up by an enquiry into the major gynaecological symptoms:

1. Pain, bleeding (relation to intercourse), discharge or pruritus.
2. Sensation of prolapse, any urinary tract or bowel symptoms.
3. Infertility, sexual function, marriage.

Often you will not uncover any problems in these areas unless you ask about them specifically.

Past Health
1. Menstrual history:
   a. Age at menarche.
   b. Regularity and duration of period.
   c. Nature of period – how heavy (how many pads, etc.) what is the colour, are there any clots.
2. Obstetrical history.
3. Contraceptive history.
4. Medical and surgical history:- ask about D & C's specifically.

- Last pap smear

Family Health — Ask about close relatives, and any specific conditions that are indicated (e.g. thyroid disorders if menorrhagic or amenorrhagic, diabetes if infertile).

Social History — Occupation. Is V.D. a possibility in her case.
Financial security.
Social adequacy, etc.

System Review

Ask about:	Medications and drugs at present or recently (i.e. during present illness). Allergies. Smoking. Alcohol intake. Weight. Last Pap. smear.

Then investigate systems in detail as indicated by findings.

Gynaecological Examination

General impression	Note anything obvious. Note degree of sexual development.
Hands and nails	
Radial pulse,	Blood pessure
Feet and legs	Swelling, varicosities.
Face and oral cavity	
Neck	JVP, thyroid.
Thorax	Inspect Auscultate the heart and lung fields
Breasts	Inspect Palpate for lumps
Abdomen	Inspect Palpate
Vagina	Speculum examination Digital examination *N.B.* If cystolcele, rectocele or stress incontinence is suspected, ask the patient to cough before withdrawing your fingers and determine whether an impulse is felt. Describe findings systematically as set out under obstetric examination.

Rectal examination if indicated.

Ancilliary Investigations

Routine Pap. smear

Others as indicated

CHAPTER 2

NORMAL PREGNANCY AND NORMAL PUERPERIUM

General Instructional Objective

Understands the changes in normal pregnancy and puerperium so that he can manage a normal pregnancy and puerperium.

Specific Behaviours

1. Explains the significant signs of pregnancy.
2. Explains changes in the puerperal patient.
3. Discusses investigations performed in normal pregnancy.
4. Explains the changes due to pregnancy.
5. Demonstrates ability to manage normal pregnancy and normal puerperium.
6. Discusses the pharmacology of and indications for drugs used in normal pregnancy and puerperium.
7. Demonstrates empathy and emotional support to patient during pregnancy and puerperium.

Normal Pregnancy and Puerperium

A. The Symptoms And Signs Of Pregnancy

The significant signs of pregnancy are expressed in a summarised form in Table 2.1.

1. The basal body temperature chart (see Fig. 16.7). If a patient had been keeping a temperature chart, the persistence of the raised temperature after ovulation, together with absence of menstruation, is the earliest sign of pregnancy. This is due to the persistence of progesterone secretion from the corpus luteum.

2. Pregnancy test All tests depend on the production of chorionic gonadotrophin.

a. *Biological tests:*

i. Ascheim-Zondek Test – This is positive if haemorrhagic follicles or corpora lutea develop in a female mouse 5 days after injection of the patient's urine.

ii. Friedman test – As above, but here a female rabbit is used and results are obtained in 2 days.

iii. Hogben test – Injection of the pregnant patient's urine into a female toad cause causes ovulation with visible release of ova within 12 to 14 hours.

b. *Immunological tests:*

Several agglutination inhibition tests exist which are based on the principle illustrated in Figure 2.1. The tests differ in their agglutinating particles which may be latex particles (Gravindex), or red blood cells (Prognosticon; Prepuerin). The test using red blood cells is more accurate (98%) than that using latex (92%), but the former takes some 2 hours to read while the latter only 2 minutes.

Performed early (e.g. 8 days after a "missed period") the test may not react and a confirmatory test is usually required at 6 weeks amenorrhoea. False positives may be obtained during a mid-cycle L.H. peak (H.C.G. and L.H. cross-react), or premenopausally when L.H. rises.

3. Amenorrhoea – In a previously menstruating woman the presence of amenorrhoea suggests that a patient is pregnant until proven otherwise. Pregnancy may, however, be present in spite of menstrual-like bleeding whose mechanism is not known, or due to implantation (placental sign).

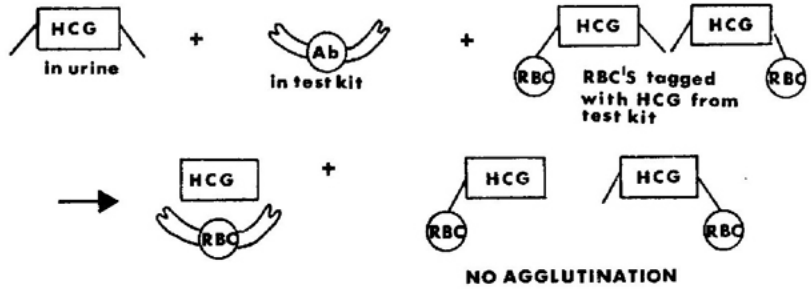

Fig. 2.1. Principle of the agglutination inhibition test. A positive pregnancy test is illustrated in which agglutination of red blood cells was *inhibited* by the presence of urinary HCG that bound the antibodies from the test kit.

Symptoms and Signs of Pregnancy

	Weeks Amenorrhoea	*Significant Signs or Symptoms*
1.	2-3	Basal body temperature chart
2.	5-6	Pregnancy test
3.	4	Amenorrhoea
4.	4-9	Morning sickness
5.		Breast changes
6.	6-14	Bladder changes
7.		Cervical changes
8.	6-8	Palpable uterine changes
9.		Vaginal and vulval changes
10.		Hegar's sign
11.		Uterus palpable abdominally
12.		Ultrasound echoscopy
13.		Ballottement
14.	16	X-ray signs
15.		Quickening (multigravida)
16.	16	Abdominal enlargement
17.		Quickening (primigravida)
18.		Palpable uterine contraction
19.		Palpable foetal movements
20.		Audible foetal heart sounds
21.		Palpable foetal parts.

TABLE 2.1 (Modified from Garrey *et al*, 1972.)

4. Morning sickness. This occurs in 50 to 60% of pregnant women between the fourth and ninth weeks of pregnancy, although it may persist longer. Suggested causes include raised levels of circulating human chorionic gonadotrophin and oestrogens. Morning sickness is usually adequately relieved by Debendox, but where antiemetic and tranquilizer effects are required Stelazine is prescribed. (See page 29)

5. Breast changes. Increased vascularity, a sensation of heaviness and almost of pain is due to the raised oestrogen and progesterone levels. There is also an increase in the pigmentation of the nipple and areola, together with enlargement of the areola. Montgomery's tubercles (intermediates between sweat and true mammary glands) become prominent. By week 16 colostrum may be expressed from the breast.

6. Bladder symptoms. Frequency will occur between the 6th and 14th weeks amenorrhoea initially. Its causes include an increased renal glomerular filtration rate, bladder hyperaemia, and pressure of the uterus on the bladder.
Frequency late in pregnancy (36 to 40 weeks) is caused by the compression of the bladder by the uterus against the pelvic brim.

7. Cervical changes consist of softening and a deep blue discoloration of the cervix, due to increased vascularity and water content, and reduced collagen.

8. Palpable uterine enlargement is best compared with everyday objects:

- golf ball size – 6 weeks
- small lemon – 6 to 8 weeks
- orange – 8 to 10 weeks
- grapefruit – 12 to 14 weeks

9. Vaginal and vulval changes include pulsation in the lateral fornices, darkening of the vaginal mucosa, and vulval varicosities (Kluge's sign).

10. Hegar's sign is elicited on bimanual examination when, due to the softening of the lower segment by oestrogens the examiner's fingers appear to almost meet (Fig. 2.2.) and is usually felt between 8 and 10 weeks.

11. The uterus is palpable abdominally at about 12 weeks amenorrhoea. It is felt as a definite mass above and deep to the symphysis pubis.

12. Ultrasonic echoscopy can be used to visualise the uterine contents.

13. Internal ballottement is illustrated in Figure 2.3. and it depends on a freely floating foetus within the membranes.

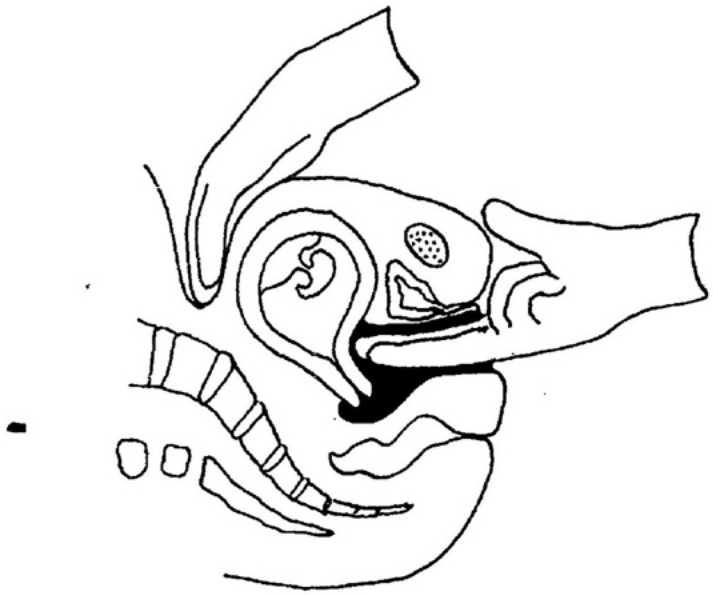

Fig. 2.2. Hegar's sign. The examiner's fingers appear almost to meet.

14. X-ray signs. With good techniques calcification of foetal bones may be seen at 16 weeks.

15. Quickening in the multigravida is probably felt earlier than in the primigravida because of previous experience.

16. Abdominal enlargement is first noticeable at about 16 weeks.

17. Quickening in the primigravida should be enquired for. The feeling of early foetal movements may be described as a faint "butterfly" movement within, or that of "bubbles" in the abdomen.

18. Palpable uterine contractions. Soon after implantation the uterus exhibits intermittent, painless, irregular contractions. These become palpable at about 20 weeks, and are called Braxton Hicks' contractions.

19. Palpable foetal movements. Detection of these is irregular and may depend on the thickness of the abdominal wall.

20. Audible foetal heart sounds. This refers to the foetal heart sounds that are picked up in the antenatal clinic with a foetoscope. These appear at about 24 weeks amenorrhoea. With electronic means the heart beat can be picked up much earlier.

21. Palpable foetal parts. This sign is used more for finding the

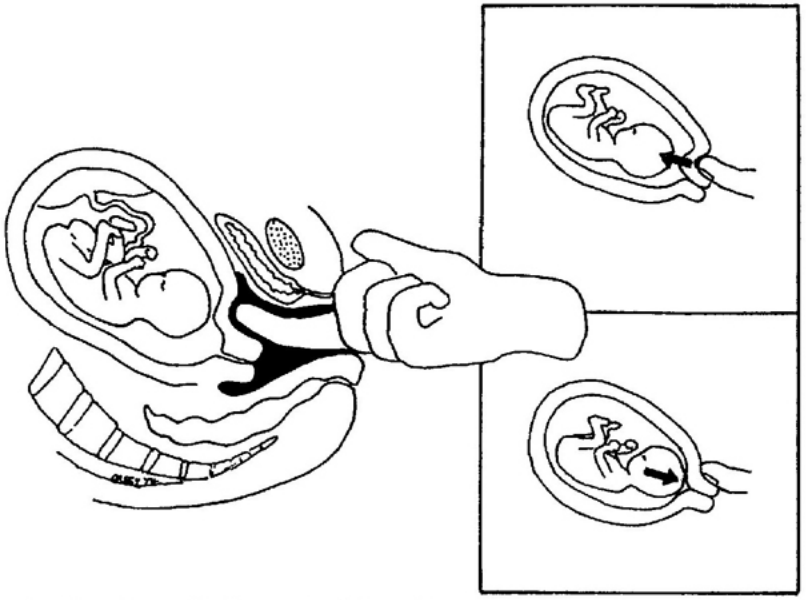

Fig. 2.3. Internal ballotement of feotus in early pregnancy.

lie and presentation than to confirm the presence of a pregnancy.

B. Physiological Changes In Pregnancy

In the pregnant woman not only does the genital tract show changes but physiological alteration take place throughout the body.
Three generalisations may be made with respect to *central nervous system.* The patient may exhibit easy fatigability and somnolence, euphoria and well-being, and sometimes depression and chronic fatigue probably due to excessive weight carrying.

In the *cardiovascular system* the cardiac output increases by a maximum of 20% due to both rate and stroke volume increase. The cause of this is obscure but may be due to the placenta acting as a shunt. The peripheral vessels reduce their resistance so that peripheral blood flow increases 6-fold near term. Progesterone reduces vascular muscle tone, and oestrogen reduces the acid mucopolysaccharides in the walls of the larger vessels. Varicosities in the legs are more common due, in part, to the higher venous pressure distal to the uterine obstruction of the inferior vena cava.

Haematological changes include a blood volume increase of some 50% by the end of the second trimester, mainly due to an increase in the plasma volume as a result of aldosterone antagonism by progesterone and water retention by oestrogens (Fig. 2.4.) The haemoglobin falls as a consequence of the plasma volume increase, to some 12 g % at 32 weeks, even though the total red blood cell mass does show some increase (Fig. 2.4.) The leucocyte count increases from a mean 4500 to 7500 cells/mm^3, and platelets rise from some 200,000 to 300,000 near term, and 600,000 in the puerperium. The erythrocyte sedimentation rate (ESR) rises four-fold with the increased levels of fibrinogen. Haemodilution masks the increase in the total plasma proteins. The more important changes are shown in Table 2.2. Blood lipid levels also rise significantly, being highest near term.

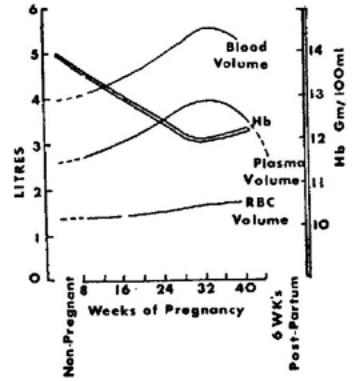

Fig. 2.4. Blood volume and haemoglobin changes in the normal pregnancy. (Modified from Hytten *et al,* 1963.)

Changes in the *respiratory system* include an increase in the tidal volume (minute volume rises from 7.25 to 10.5 litres) due to a 20% rise in the oxygen consumption. Near term the lower ribs tend to flare out.

Rising oestrogens are responsible for the gum hypertrophy sometimes found in pregnancy. Progesterone, by reducing muscle excitability decreases intestinal motility leading to constipation. The musculature of the cardiac sphincter is relaxed and may allow regurgitation and heartburn.

Renal blood flow increases by 25 to 50% and the glomerular filtration rate by some 50%. However, since tubular reabsorption is unaltered the clearance of many solutes for example, urea, uric acid, and glucose, is increased.

Metabolic changes in pregnancy are complex and under much investi-

gation. There is a general increase in the metabolic rate, largely due to foetal demands. Oxygen consumption rises by 20% and the thyroid gland hypertrophies in perhaps 70% of patients.

The carbohydrate metabolism is affected by human placental lactogen during pregnancy. This hormone antagonize the action of insulin, breaks down body fat, and thus acts towards the elevation of blood glucose levels. As a result insulin rises to even higher levels increasing glucose utilisation but restricting any abnormal blood levels. (Fig. 2.5). The increased demand on the pancreas may at this stage uncover a latent diabetic.

Protein metabolism shows an overall positive nitrogen balance, some 500 grams of protein being retained by term. This fact calls for a high protein diet during pregnancy.

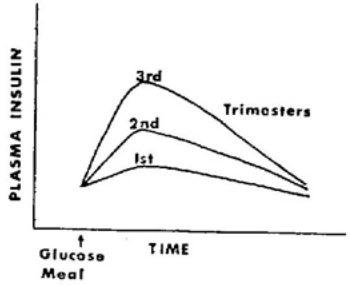

Fig. 2.5. Plasma insulin response to a glucose test meal Pancreatic stimulation by glucose is maximal during the third trimester due to steadily rising levels of the insulin inhibitor, placental lactogen. Modified from Hytten et al, 1971.

Fat is the main form of maternal stored energy during pregnancy and most of it is in the form of depot fat. Blood lipid also increases significantly. The importance of this lies in the fact that since glycogen stores are low any major stress will draw quickly on fat for energy thus predisposing to ketosis.

The average total weight gain should be some 12.5 kg (28 lbs.), the main increase being in the second half of the pregnancy. It should not exceed 500gm/week.

Endocrine changes in pregnancy are numerous and important. Sophisticated immunoassay techniques have revealed an unexpected versatility of the placenta as a source of pituitary-like hormones.

a. *Human Chorionic Gonadotrophin* (HCG).

HCG is a glycoprotein with a molecular weight of about

30,000, and is produced by the trophoblast. It is tempting to conclude that Langhans cells of the cytotrophoblast are the ultimate source since their decline in the placenta parallels the production of HCG (Fig. 2.6, Hytten *et al*, 1971). Radioimmunoassay techniques can detect HCG in the serum 10 days after ovulation or 2 to 3 days after implantation, but standard pregnancy tests become reliable some 26 days after conception, or about 40 days after the last menstrual period (L.M.P.) in a woman with 28 days cycles. The action of H.C.G. is to maintain the corpus luteal secretion of oestrogen and progresterone until the placenta has developed sufficiently to take over all steroid production.

High levels of H.C.G. may be associated with:

. Hydatidiform mole
. Choriocarcinoma
. (Choriocarcinoma of the testis)
. Twins
. Severe pre-eclamsia (Leraine *et al*, 1971)

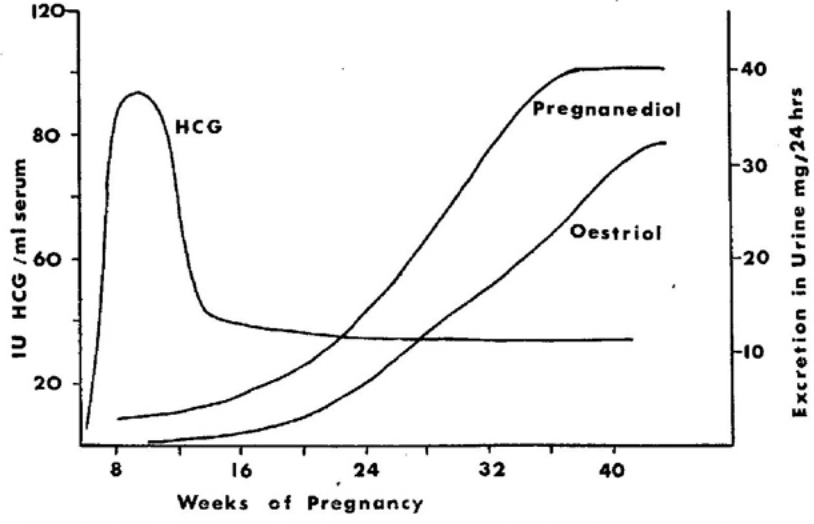

Fig. 2.6. Serum chorionic gonadotrophin and urinary pregnanediol and oestriol patterns in normal pregnancy. Modified from Hytten et al, 1971.

b. *Human Placental Lactogren* (H.P.L)

This hormone was previously called human chorionic somatomammotrophin (H.C.S.). Produced by the trophoblast

it rises steadily throughout the pregnancy (Fig. 2.7). Levels of H.P.L. can therefore be used to determine the state of the placenta, although this test is not generally used as yet, being in an experimental stage. H.P.L. has a mammotrophic effect on the growth of the breast, and may influence also carbohydrate and lipid metabolism.

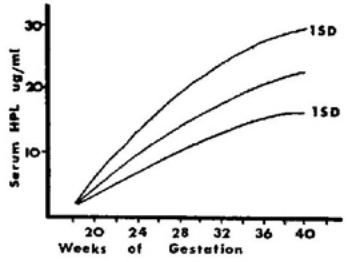

Fig. 2.7. Serum placental lactogen in normal pregnancy. Modified from Saxena et al, 1968.

c. *Oestrogen and Progesterone*

During the first 12 weeks of gestation ovarian (corpus luteum) production of oestrogen and progesterone is important in maintaining the pregnancy. After this time placental production of both hormones makes the foetus more self-sufficient. In the production of oestrogen the foetus and placenta work together as a unit, in which the placenta can carry out certain synthetic steps, and the foetal adrenal and liver the remaining steps. Measurement of the urinary levels of oestriol, a degradation product of the potent 17 oestradiol can therefore give an index of the well-being of the foetoplacental metabolic unit. The syncytial cell of the trophoblast is probably the ultimate source of the hormone (Deane *et al*, 1953).

Actions of Oestrogens During Pregnancy

i. *Protein synthesis* is stimulated at the cellular level.
ii. *Uterus* – oestrogen stimulates growth of the myometrium.
iii. *Breast* – growth of the duct system is stimulated.
iv. *Connective tissue* – alternation of polymerization of acid mucopolysaccharides increases the stretch properties of collagen (Hytten *et al.* 1971), and increases hygroscopic qualities producing water retention.
v. *Serum protein changes* – there is an increase in hepatic protein production (see Table 2.2).

Plasma Protein Alterations During Pregnancy

Protein		*Change*
Total protein	–	Rises
Total protein concentration	–	Falls (due to haemodilution.)
Albumin : Globulin ratio	–	Falls
Albumin alpha, alpha 2 and beta	–	Falls
Globulins : α, $\alpha 2$, and β	–	Rise (transport globulins)
Gamma globulins		
Gamma globulins	–	No change
Fibrinogen	–	Rises (25 to 50% increase).
Clotting factors 7,9,10,12	–	Rise

Table 2.2

Actions of Progesterone During Pregnancy

i. *Reduction of smooth muscle excitability* especially in the uterus, protects the foetus from expulsion. To a lesser degree this same effect is seen in the ureters, stomach, and large bowel, predisposing these to dilatation and reduced motility.
ii. *Hyperthermia* – progesterone causes the raised basal temperature after ovulation.
iii. *Fat metabolism* – progesterone promotes fat storage.
iv. *Breast* – growth of alveolar structures is stimulated.

One ought to keep in mind that many of the abovementioned changes are to some extent dependent on more than one hormone.

d. *Other Hormones* Adrenal cortical homones, thyroid hormones, and numerous other hormones undergo various changes during a pregnancy, but these will not be discussed here. For detailed descriptions refer to Hytten *et al*, 1971.

C. Investigations Performed in The Normal Pregnancy

1. *Pregnancy test:* This is performed on an early morning midstream urine specimen (see page 2.1) (only if there is doubt regarding the fact of the pregnancy).

2. *Blood tests:* Performed at the first visit.

   a. *Blood group.* The ABO and Rh groupings are performed.
   b. *Antibody screening. All* patients whether Rh +ve or –ve, are tested. Besides D-antibodies occasional anti-c, anti-Kell, and immune anti-A and anti-B antibodies will be detected.

The *purpose* here is to forewarn any cross-matching difficulties, and to *alert* the doctor to such babies as may need exchange transfusions. In Rh —ve women screening should be carried out again at 28 weeks and if D-antibodies are found appropriate management is undertaken (see Chapter 8). At any stage, the *mere presence of D-antibodies is significant* no matter what the titre.

c. *Haemoglobin* – At about 12 weeks the lower limit is 12·0gm% At no time during the pregnancy should the haemoglobin fall below 10·5gm%. A second haemoglobin estimation is made at 32 to 34 weeks.
d. *Rubella antibody titre.* A titre of 1/20 or more is positive evidence of rubella infection. The test provides a baseline for any future comparison if infection is suspected.
e. *Wasserman* (complement fixation) and Kahn (flocculation) tests for syphilis are routinely carried out.

3. *Urinalysis:* A mid-stream specimen is tested after a vaginal toilet to remove contaminating secretions.

   a. *Side-room analysis* – including specific gravity, protein and sugar content are performed.
   b. *Bacilluria screen test* (B.S.T.) is routine in some centres, and should be carried out when urinary tract infection is suspected.

4. *Papanicolaou smear.* A cervical smear for cancer is routine on the first visit in antenatal clinic.

5. *Chest X-ray* – is ordered if a year has elapsed from a previous chest Xray.

D. Management of a Normal Pregnancy

After confirmation of the pregnancy, enrol the patient in an *antenatal clinic* (whether private or hospital) and explain the importance of attending regularly (see Chapter 8). For an accurate assessment of the early uterine size it is essential to have the patient attend an antenatal clinic before 12 weeks amenorrhoea.

During the first visit:

1. *Full History*

   Record the date of the last normal menstrual period, and calculate the expected delivery date (EDD).
   Take a full medical history enquiring especially about past rubella infection, diabetes, renal disease, cardiac function, respiratory function, and any past illnesses.
   Take a full surgical history including any previous blood transfusions, surgery on the genital tract (previous Casesarian section), and abdominal surgery.
   Record in detail any previous obstetric and gynaecologic history (see Chapter 1) emphasizing any previous abnormal conditions.

2. *Examination*

   A general and pelvic examination is carried out (see Chapter 1). During the speculum examination a cervical smear is taken. On bimanual palpation the size of the uterus is estimated.

3. *Laboratory Investigations*
   Routine tests described above are performed.

Subsequent Visits:

a. 4th weekly — until 32 weeks
b. Fortnightly — until 36 weeks
c. Weekly — until delivery

During the "subsequent visits" a routine is followed which will include:

- General enquiries as to health, especially any discharges, pain, or bleeding. Any questions are answered.
- Weight, blood pressure, fundal height, presence of foetal heart sounds or of movements.
- Oedema of the hands.
- A urine sample is tested for protein and sugar.

At 32 to 34 weeks the foetal position is checked and external version may be undertaken in case of a breech presentation. At this visit the second haemoglobin estimation is made.
At 36 to 38 weeks a pelvic examination should ideally be carried out to assess pelvic adequacy.

Counselling

It is the obstetrician's duty to volunteer information to the pregnant patient concerning intercourse, clothing, exercise, immunization, smoking and travelling. Various misconceptions concerning, for

example, diet, should be cleared up. Unmarried mothers may obtain a government allowance and this information should be supplied.

E. Changes In The Puerperal Patient

The *puerperium* may be defined as that period following childbirth in which the genital organs return to their pre-pregnant condition. It is usually regarded as being 6 weeks following delivery.

1. *The Uterus.* At the end of the third stage of labour the size of the uterus is that of a 20 week pregnancy. Over the next six weeks it's weight reduces from 1000g to some 40g, and remains slighty larger than in the pre-pregnant condition. This involution takes place by cytoplasmic autolysis secondary to oestrogen withdrawl. Clinically the uterus regresses by about one finger-breadth per day, sinking behind the symphysis by day 10 to 12 postpartum.
2. *Endometrium* regenerates by day 10 postpartum in all areas except at the placental site. Here regeneration takes some 6 weeks.
3. *Lochia* is the puerperal vaginal discharge. Initially, for about 4 days, a red vaginal discharge (lochia rubra) is present, but this is soon replaced by a more serous one (lochia serosa) for the next 6 days or so. Finally, for the next 4 weeks a yellowish to white discharge (lochia alba) is produced which diminishes in volume and disappears.
4. *Cervix.* The cervix is at first flabby, bruised, purple, and will admit 2–3 fingers. Within 7 days due to water loss and muscle growth the os will admit one finger only. The permanent change in the cervical os is illustrated in Figure 2.8.

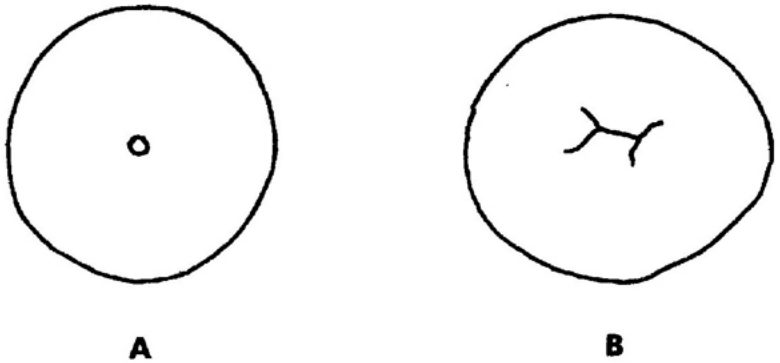

Fig. 2.8. The cervical os in the A. nulliparous, and B. parious woman

5. *Vagina and Vulva* rapidly regain their tonicity and by week 3 after parturition the vaginal size has returned to normal.
6. *The Blood* changes include a reduction in the volume and cardiac output, which reach normal levels by day 5. The haemoglobin falls. There is a leucocytosis at parturition which slowly resolves. The fibrinogen levels increase in week one after parturition and then fall to normal levels.
7. *Weight* loss on the average is 2.5 kg (5½ lbs.) during the first week after delivery.
8. *Urinary tract.* Acute retention of urine may follow parturition due to a reflex suppression of micturition, sphincter spasm, and oedema and hyperaemia of the bladder. This may last for up to 24 hours and catheterization will need to be performed. During the days following a diuresis takes place. By two weeks after parturition the dilated ureters and pelves are nearing their normal size.
9. *Breast* changes are described later under 'lactation'.
10. *Re-establishment of Menstruation* is variable but usually takes some 10–12 weeks in the non-breast feeding mother, and 14 or more weeks in the breast feeding mother.

F. Management of The Normal Puerperium

The management will be discussed under a number of headings.

1. *Rest* and sleep is the most important need after a labour. Often an analgesic will be required.

   Meanwhile a 4-hourly temperature chart is commenced to detect any infection or other complications.
2. *Early ambulation* helps to strengthen the pelvic floor muscles and to drain the lochia. Advice on post-natal exercises is also given.
3. *Toilet of vulva.* A sterile pad is worn as long as lochia discharges, and a shower is taken after each motion.
4. *Rooming-in* with the baby during the day is the ideal situation where the facilities allow. This procedure is psychologically good since it reduces anxiety, and allows demand-feeding to be practised as well as giving mother the opportunity to learn baby care early and under supervision.
5. *"Third day blues".* Temporary depression coinciding with breast

engorgement may set in, when the excitement of the birth has died down and problems of reality are returning. Sympathy and counselling are required.

6. *After pains* occur especially in the multipara after breast-feeding, and are due to uterine involution contractions as stimulated by oxytocin (Fig. 2.9).

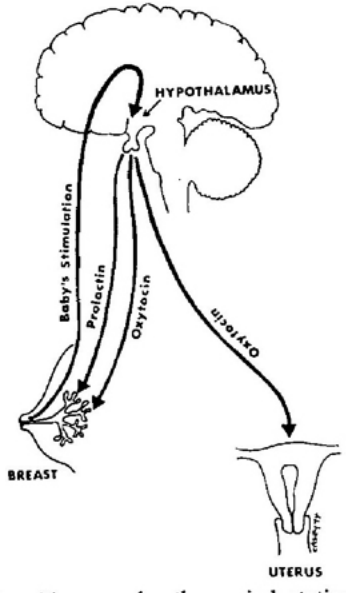

Fig. 2.9. The neural and hormonal pathways in lactation. The baby's stimulation of the nipple sends neural impulses to the hypothalamus where oxytocin is produced. Oxytocin release by the posterior pituitary leads to the movement of milk in the breast from alveoli into ducts, as well as an acceleration of uterine involution. Prolactin release from the anterior pituitary leads to the mobilization of raw materials for milk production.

7. *Discharge from hospital* is effected when:

   . the baby is feeding well, and is not jaundiced,
   . the uterus is involuting normally,
   . all tears and incisions are healing,
   . there is no suspicion of deep venous thrombosis,
   . urinary function is normal.

   A post-natal visit is arranged for 6 weeks after parturition, meanwhile the patient is warned to report any fever, bleeding, or breast tenderness.

During the *post-natal visit* attention is directed at the return of the genital tract to normal, at lactation, and at any problems that the patient may have, such as stress incontinence. A cervical smear is also taken. Discuss contraceptive advice and offer the various alternatives.

G. Lactation and Breast Changes

During pregnancy the effect of oestrogens and progesterone on the breast is to increase the growth of alveoli and terminal ducts, increase the weight of the breast, and cause pigmentation, venous engorgement and prominence of Montgomery's tubercle. The stage is set for milk production but none is produced, except small amounts of colostrum, until the oestrogens and progesterone production falls at parturition. Milk "comes in" at about the third day post-partum. The mechanism of lactation is illustrated in Figure 2.9. The baby's stimulation of the nipple sends neural stimuli to the hypothalamus which releases oxytocin and mediates the release of prolactin. The prolactin mobilizes the raw materials required for milk production. The oxytocin causes the myo-epithelial cells in the breast to contract forcing milk out into the duct system of the breast. It also speeds up uterine involutions. The amount of nipple stimulation therefore determines the amount of milk produced. The baby does not suck the milk out but rather compresses the lactiferous ducts with its gums so forcing the milk into its mouth. The maximum time in minutes, spent by the baby at each breast, should be no greater than the baby's age in days, reaching a limit of 10 minutes.

Advantages of Breast Milk and Breast Feeding

When comparing breast milk with cow's milk the following advantages may be observed:

Protein: Even when diluted 1 : 1 cow's milk has more protein (casein) than has breast milk. The difference however, lies in the quality, the human protein being more easily digestible. Moreover, human casein promotes greater Ca^{++} and Fe^{++} absorption as well as greater sulphur retention. Maternal antibodies (IgA) are also passively transferred in the milk.

Carbohydrates: There is more carbohydrate in human milk.

Quality of fat: Although the quantity is not different, human milk has the advantage of containing the cis-cis form of the essential fatty acid, linoleic acid. Cow's milk contains this acid but in the cis-trans

form which cannot act as an essential fatty acid. Human milk also contains a higher proportion of unsaturated fatty acids.

Electrolyte load is important in the neonate since the immature kidney cannot produce concentrated urine. Even when diluted 1:1 cow's milk carries a higher elecrolyte load than human milk, and because more water is required to clear this load the infant would be more susceptible to dehydration.

Vitamins: Cow's milk contains less vitamin A, C, and E.

Susceptibility to infection is lower in the breast-fed child, in part due to the vigorous growth of lactobacillus in the lower gut, so preventing pathogens from gaining a foothold. Human milk is rich in oligosaccarides which encourage growth of lactobacillus bifidus.

Other advantages: From the maternal point of view the following advantages are probably the most important.

. An emotional bond and maternal satisfaction develops. This obviously is difficult to measure but is a reality to those who have experienced the feeling.
. Uterine involution speeds up (Fig. 2.9).
. Menstruation is inhibited for a longer period of time probably as a result of prolactin's inhibition of F.S.H. and L.H. release.
. Breast milk is always at the right temperature, it is free, and there is no need for sterilizing, mixing, heating, bottle washing, or special arrangements when travelling.

Complications of Lactation

1. *Inadequate lactation* is usually due to inadequate prolactin production through lack of nipple stimulation (Fig. 2.9), and here intervals between feeds should be reduced to 3 hours.

2. *Breast engorgement* results from a failure of the "let-down" reflex. It may be improved by sub-lingual or intranasal oxytocin, together with manual expression, if the baby cannot suckle.

3. *Infection* is predisposed to by lack of hygiene, obstruction and stasis, and cracked nipples. Since the hospital staphylococci are usually penicillin resistant streptomycin by injection is the drug of choice. Culture of milk for drug sensitivity is also done.

4. *Cracked nipples* result from long periods of suckling. Treatment consists of taking the baby off the breast, while using manually expressed and sterilized milk for feeding. A neomycin and bacitracin containing ointment may be applied.

Suppression of Lactation

After a stillbirth or when the baby is for adoption, lactation is suppressed. Although stilbestrol is still used by many, the risk of thrombosis is high in the puerperium and it is probably better to suppress lactation by avoidance of breast stimulation, with expression of the milk when the breasts are tender.

H. Pharmacology and Indications for Drugs used in Normal Pregnancy

1. *Iron and Folic acid:* FEFOL 2 SPANSULE (NHS-30 plus 2 repeats).

   . *Contains* 270 mg of ferrous sulphate and 300 micrograms of folic acid.
   . *Indicated* in prophylaxis of iron and folate deficiency anaemia of pregnancy, one tablet is taken nightly throughout pregnancy. Gastric irritation is unlikely.

2. *Antiemetic agents:*

   Trifluoperazine: STELAZINE (I mgm. b.d.)

   . *Contains* trifluoperazine, a phenothiazine.
   . *Indicated* in pregnancy if antiemetic and tranquilliser effect is necessary. Dose is 1 to 2 mg twice daily.
   . For *side effects* refer to manufacturer's detailed information.

   Dicyclomine: HCl. (10 mgm. 2 each night)

   . *Contains* an antispasmodic, and antihistamine, and pyridoxine.
   . *Indicated* in nausea and vomiting of pregnancy (morning sickness), two tablets being taken before retiring at night. In more severe cases a morning tablet may be taken as well.
   . *Side effects* may include atropine-like effects, and drowsiness.

3. *Calcium* supplement is needed only if the patient is not drinking milk daily.

4. Constipation, which becomes a common problem to women should be treated by giving a mild laxative such as METAMUCIL.

5. During pregnancy occasional diarrhoea can be easily controlled with LOMOTIL (2 tablets every 4-6 hours).

6. Rubella immunization: No proof is available but immunization with the attenuated live virus is probably best avoided during pregnancy. In the puerperium or before a pregnancy, women having no evidence of past German measles should be vaccinated with CENDEVAX.

Drugs Adversely Affecting the Human Foetus

During any pregnancy only absolutely necessary drugs should be prescribed. The following list deals with some of the more dangerous drugs. The stage of pregnancy at which maximal damage results is indicated in each group.

a. *Masculinizing drugs* (1st and 2nd trimester) for example, androgenic steroids, may cause genital tract abnormalities.

b. *Stilbestrol.* An adenocarcinoma of the vagina has been noted to occur in children of mothers treated with stilbestrol during pregnancy (Herbst *et al*, 1971).

c. *Corticosteroids* are possibly associated with cleft palate malformations if given in the first trimester.

d. *Cytotoxic agents* (1st trimester) for example, methotrexate, used for control of psoriasis in the U.S.A., may produce malformations and abortion.

e. *Thalidomide* (1st trimester) is a well known example of a teratogenic drug, causing malformations in limbs, eyes, ears, and the heart.

f. *Goitrogens and Antithyroid Drugs* (throughout pregnancy) include iodine, iodine-containing radiographic media, thioureas, and perchlorates. Neonatal goitre and hypothyroidism leading to cretinism or death may occur.

g. *Quinine* (1st trimester) may result in the birth of a deaf child.

h. *Tetracyclines* (3rd trimester) stain teeth, and retard growth at the epiphyses.

i. *Coumarin anticoagulants* (throughout pregnancy) may cause haemorrhage and intrauterine death.

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CHAPTER 3

LABOUR

General Instructional Objective

Utilises obstetric skills and knowledge to assist in the management of a normal labour.

Specific Behaviours

1. Describes what is a normal labour.
2. Explains the mechanisms and progression of labour.
3. Identifies the normality and progression of labour.
4. Demonstrates sympathy, empathy, support and praise for the patient.
5. Identifies and satisfies the physical needs of the woman in labour.
6. Explains the pharmacology of and indications for the common drugs used in normal labour.
7. Demonstrates normal delivery techniques in labour ward.
8. Demonstrates an ability to assess the maternal condition during labour and following delivery.
9. Demonstrates an ability to assess the baby following delivery.
10. Identifies signs of separation of placenta, and assists in its delivery.
11. Demonstrates an ability to examine a placenta.

Labour

1. Definitions.
2. The Stages of Labour.
3. The First Stage of Labour.
4. The Second Stage of Labour.
5. The Third Stage of Labour.
6. The Physiology of Labour I: The Passages.
7. The Physiology of Labour II : The Passenger.
8. The Physiology of Labour III : The Powers.
9. The Mechanism of Normal Labour.
10. The Management of Normal Labour.
11. Care and Assessment of Neonate.

Definitions

1. *Labour* – is the process by which a foetus of at least 20 weeks gestation is expelled from the uterus.

2. *Normal Labour* (Eutocia) – a labour at term where the foetus presents by the vertex and whose delivery is followed by the placenta and is accomplished within 24 hours, without artificial aid or complication.

The Stages of Labour – Summary

Stage 1 – The stage of cervical dilatation.
Stage 2 – The stage of expulsion of the foetus.
Stage 3 – The delivery of the placenta.
Stage 4 – A fourth stage is sometimes described as that time from delivery of the placenta till the patient's condition enables transfer to the ward.

The First Stage of Labour

1. *Onset* – when the cervix starts to dilate. The exact time cannot be determined clinically, but is approximated by the onset of *painful* uterine contractions.

2. *Completion* – when the cervix is fully dilated. Classically the membranes rupture at the end of the first stage of labour – this frequently occurs during or even before the first stage.

3. *Duration* – depends on parity. In a primigravida it lasts, on average, for 12 hours. In a multi, 7 hours.

4. *Cause of Onset of Labour* – is unknown. Theories suggested include –

   a. Intrauterine tension.
   b. Production of a particular hormone – ACTH, cortisol, prostaglandin or oxytocin.
   c. Decrease in production of progesterone (Turnbull *et al*, 1974).
   d. Pressure of the presenting part on the lower uterine segment and cervix.
   e. Emotional or physical factors.

5. **Mechanism of Dilatation of Cervix –**
   a. Contraction and retraction (permanent shortening of uterine muscle fibres causes effacement - taking-up) and dilatation of the cervix and lower uterine segment. Consequently the lower uterine segment becomes thinner and the upper segment thicker.
   b. In primigravid patients effacement precedes dilatation (Fig. 3.1), whereas these processes occur somewhat simultaneously, in the multipara. This accounts for the variation in length of the first stage.

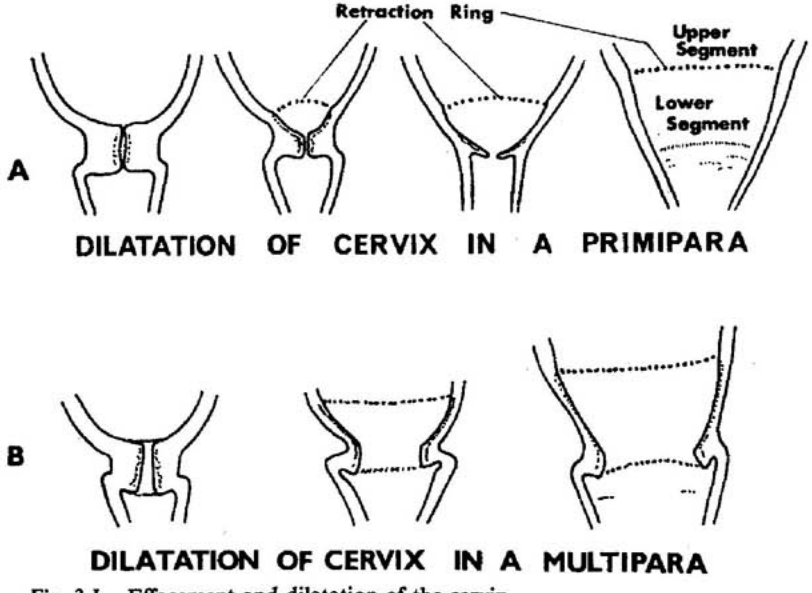

Fig. 3.1. Effacement and dilatation of the cervix.
a. in a primiparous patient
b. in a multiparous patient

c. Dilatation does not occur at a uniform rate – it is slow until the cervix is about one-third dilated, and then continues at a more rapid rate (Fig. 3.2).

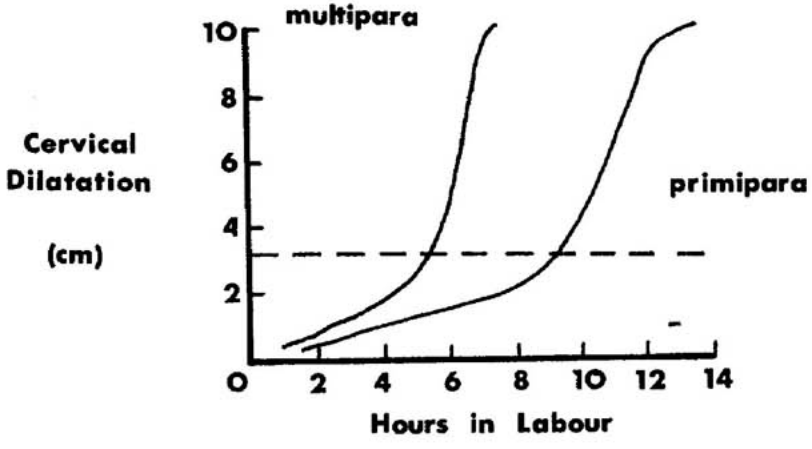

Fig. 3.2. Cervical dilatation – rate of dilatation increases when the cervix is about 1/3rd dilated.

6. **Uterine Contractions**

Frequency

a. Every 10-15 minutes in early labour.
b. Every 3-5 minutes in late first stage.

Duration

a. About one minute.
b. Reasonably constant.

Pain

a. Cervical in origin, due to resistance of the cervix to the dilating forces.

b. Occurs when intrauterine pressure exceeds 20 mmHg (once painful contractions are established), hence

c. Is slightly shorter in duration than the contraction (Fig. 3.3).

d. May occasionally be prolonged with coupled contractions.

e. Usually commences in the back and spreads anteriorly. Not invariable – may begin in the hypogastrium.

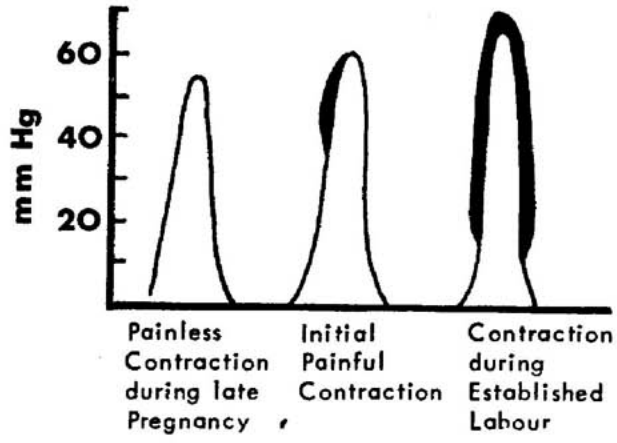

Fig. 3.3. Duration of pain is less than duration of contraction. N.B. Duration of "pain" shown by thickened line.

Origin

a. Begin at a pacemaker situated approximately at the junction of the fallopian tube and uterus.

b. One side or other is dominant.

Effect on foetus

a. Contractions gradually diminish placental blood flow, and eventually prevent it, if pressure exceeds 75mm. Hg.

b. If contractions are too long or frequent, foetal distress, may occur.

7. **"Show"** – a slight uterine haemorrhage (or passage of blood-stained mucus) due to some stripping of the membranes from the uterine wall during effacement of the cervix.

The Second Stage of Labour

1. *Onset* – can only be determined definitely by doing a vaginal examination and finding the cervix is fully dilated.
Other clinical indications include –

a. the occurrence of very painful contractions,
b. the patient desires to "bear down",
c. an increased "show",

d. the membranes may rupture,
e. a desire to defecate,
f. "physiological" haemorrhoids may become apparent.

Note: These signs are only indications, they are not infallable.

2. *Completion* – with the delivery of the infant.
3. *Duration* –
   a. In a primipara – about 1 hour.
   b. In a multipara – about ½ hour.
4. **"Bearing Down"** –
   a. This is due to descent of foetus compressing the rectum.
   b. Results in a great increase in intra-abdominal pressure which is transmitted to the uterus and reinforces uterine contractions (Fig. 3.4).
   c. Materially assists in expulsion of the foetus.
   d. Should be discouraged until the cervix is fully dilated and a contraction is occurring, otherwise, stretching of the lateral cervical ligaments occurs, predisposing to prolapse at a later stage.

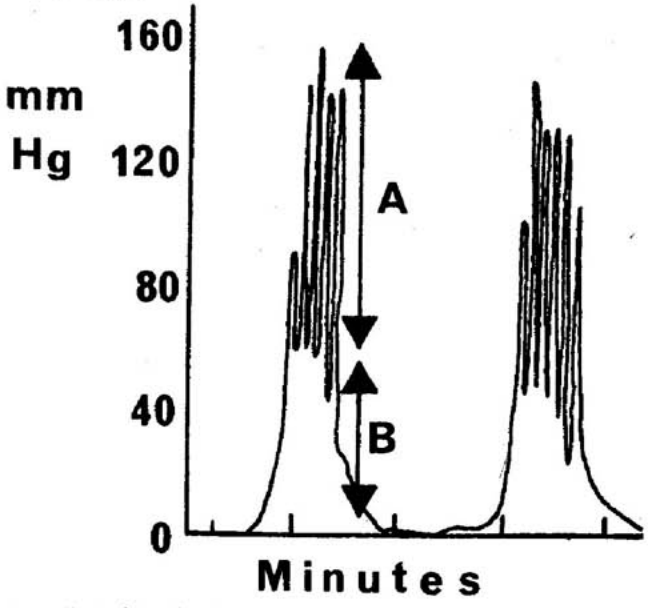

Fig. 3.4. Effect of bearing down.
A. — Increase in intrauterine pressure due to voluntary effort.
B. — Increase in intrauterine pressure due to uterine contraction.

5. **Bulging of the Perineum** –
   a. Occurs when the foetal head reaches the vaginal opening.
   b. Increases with each contraction.
   c. *May* result in perineal tearing unless an episiotomy is carried out.
6. **"Crowning"** – the term applied when the presenting part is visible, between the labia minora.
7. *Mechanism* – see section 9.

The Third Stage of Labour

1. *Onset* – following delivery of the baby.
2. *Completion* – with delivery of the placenta.
3. *Duration* – should be no more than 30 minutes.
4. *Separation of Placenta* results from –
   a. Retraction of uterine musculature (after birth of the baby), which also compresses the maternal blood vessels.
   b. Continuing contractions.
   c. Spread of extravasated blood through the decidua.
5. **Sings of Placental Separation**
   a. A permanent lengthening of the umbilical cord, outside the vulva.
   b. The fundus of the uterus rises and becomes firm and globular.
   c. A gush of blood occurs, from the vagina.
   d. Upward displacement of the fundus fails to shorten the umbilical cord outside the vulva.

The Physiology of Normal Labour I : The Passages

– the bony pelvis and the lining soft tissues.

1. *The bony Pelvis* is conveniently thought of as having a brim (inlet), a cavity and an outlet.
   a. *The pelvic brim* –
      i. *Boundaries*

. upper surface of back of symphysis pubis,
. the pubic crest,
. the pectineal eminence,
. the ilio-pectineal line (iliac portion),
. the sacroiliac joint,
. the sacral promontory.

Table 3.1 : **Summary of Pelvic Diameters**

	A-P (cms.)	Oblique (cm.)	Transverse (cms.)
Brim	11	12	13
Cavity	12	12	12
Outlet	13	12	11

Bi-ischial diameter – 10.5 cms.
Posterior sagittal diameter – 7.5 cms.

ii. *Diameters* – (q.v. Table 3.1).

. *Anteroposterior diameter* (true conjugate) – from the posterior superior margin of the pubic symphysis to the centre of the sacral promontory (11 cms)

. Two *Oblique diameters*, e.g. the right oblique extends from right sacroiliac joint to the left pectineal eminence (12 cm.)

. *Transverse diameter* – the widest part of the inlet (13 cm.) Not a true diameter as it does not pass through the centre of the inlet.

b. *The cavity* – between the pelvic inlet and outlet.

i. *The plane of greatest dimensions* – almost circular, bounded by–anteriorly, the midpoint of the back of the symphysis pubis and posteriorly, the junction of the second and third sacral vertebrae. (Fig. 3.5).

ii. *The plane of least dimensions* – the most important plane of the pelvis. It extends from the lower border of the symphysis pubis to the ischial spines and the lower border of the last sacral vertebrae (Fig. 3.5). The pelvic canal turns sharply forward at this level.

iii. *The bi-ischial diameter* – the distance between the two ischial spines.

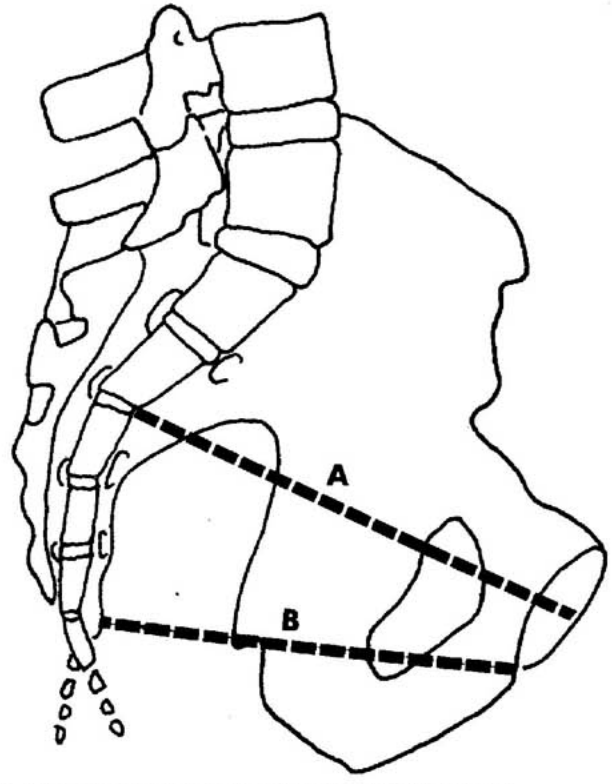

Fig. 3.5. The planes of greatest and least pelvic dimensions.
A. — Plane of greatest pelvic dimensions.
B. — Plane of least pelvic dimensions.

c. *The Outlet* – extends from the plane of least pelvic dimensions to the anatomical outlet.

i. *The anatomical outlet* – consists of two planes joining at the ischial tuberosities (Fig. 3.6). Its boundaries are –

. the lower border of the symphysis pubis,
. the ischial tuberosities, and
. the coccyx.

ii. *Diameters* –

. *Antero-posterior diameter* – from the middle of the lower border of the symphysis pubis to the tip of the last sacral vertebra (13 cms.)

TABLE 3.2

COMPARISON OF THE SHAPE, SIZE AND CAPACITY OF THE DIFFERENT TYPES OF MATERNAL PELVES (Classification of Caldwell and Malloy) AND THE EFFECT ON LABOUR

a. PELVIC INLET

	Gynaecoid	Android	Anthropoid	Platypelloid
Sex Type	Normal female	Male	Ape-like	Flat female
Incidence	40 per cent	20 per cent	35 per cent	5 per cent
Shape	Round or transverse oval. Transverse diameter is a little longer than the antero-posterior	Heart or wedge-shaped	Long antero-posterior oval	Transverse oval
Antero-Posterior Diameter	Adequate	Available diameter short	Long	Short
Transverse Diameter	Adequate	Long	Adequate,	Long

b. THE CAVITY

	Gynaecoid	Android	Anthropoid	Platypelloid
Antero-Posterior Diameter	Adequate	Reduced	Long	Adequate
Transverse	Adequate	Reduced	Adequate	Adequate
Posterior Sagittal	Adequate	Reduced	Adequate	Adequate
Anterior Sagittal	Adequate	Reduced	Adequate	Adequate
Sacrum	Wide deep curve. Short, slopes backward, light bone	Flat. Inclined forward, long, narrow, heavy	Inclined backward. Narrow, long	Wide deep curve
Sidewalls	Parallel, straight	Convergent. Funnel pelvis	Straight	Parallel
Ischial spines	Not prominent	Prominent	Variable	Variable
Sacro-sciatic Notch	Wide and short	Narrow, long, high arch.	Wide	Short
Depth: Ilio-pectineal eminence to tuberosities	Average	Long	Long	Short
Capacity	Adequate	Reduced in all diameters	Adequate	Adequate

TABLE 3.2

(continued)

c. THE PELVIC OUTLET

	Gynaecoid	Android	Anthropoid	Platypelloid
Antero-Posterior Diameter	Long	Short	Long	Short
Transverse Diameter (Bituberous)	Adequate	Narrow	Slightly below normal	Wide
Pubic Arch	Wide and round 90 degrees	Narrow and deep 70 degrees	Normal to narrow	Very wide
Inferior Pubic Rami	Short and concave inwards	Straight and long	Long and slightly curved inwards	Straight and short
Capacity	Adequate	Reduced	Adequate	Adequate

d. THE EFFECT OF LABOUR

	Gynaecoid	Android	Anthropoid	Platypelloid
Foetal Head	Engages in transverse or oblique diameter in slight asynclitism. Good flexion. OA is common.	Engages in transverse diameter Extreme moulding	Engages in antero-posterior or oblique. Often occiput posterior	Engages in transverse diameter with marked asynclitism
Labour	Good uterine function. Early and complete internal rotation. Spontaneous delivery. Wide pubic arch reduces perineal tears	Deep transverse arrest is common. Arrest as OP with failure of rotation. Delivery is often by difficult forceps application, rotation, and extraction. The narrow pubic arch leads to large perineal tears	Delivery and labour is usually easy. Birth face to pubis is common	Delay at inlet. Once the inlet is passed, labour is like that in a gynaecoid pelvis
Prognosis	Good	Poor	Good	Delay at inlet

. *Transverse diameter* – between the inner aspects of the two ischial tuberosities (11 cms.).

. *Posterior sagittal diameter* – from the midpoint of the transverse diameter to the tip of the sacrum.

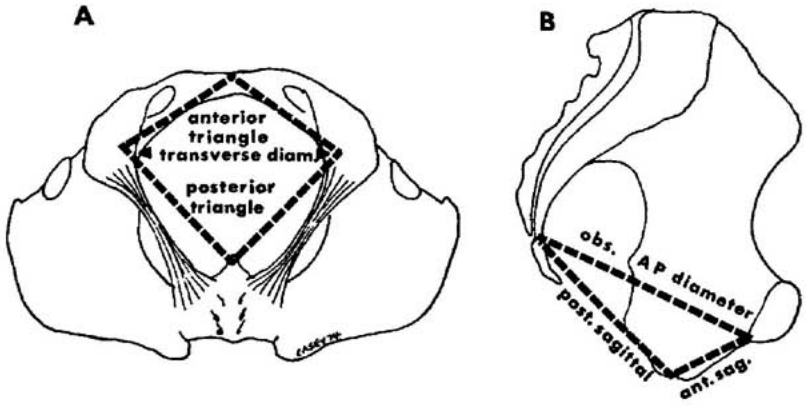

Fig. 3.6 The outlet of the pelvis:
a. from below b. sagittal section

Classification of the Pelvis:

There is a complete spectrum of shapes, planes and diameters of the female pelvis. There are four basic types – gynaecoid, android, anthropoid, and platypelloid – based on the shape of the inlet. These characteristics are described in Table 3.2. (modified from the classification of Caldwell and Malloy), and Figs. 3.7 A, B.

2. **The Soft Passages**

The *pelvic floor* comprises the soft tissues filling the pelvic outlet. It is pierced by three canals – the urethral, vaginal and rectal canals.

The major structure of the pelvic floor is the levator ani muscle. It arises from back of the body of the pubis, the tendinous arch of pelvic fascia and the pelvic aspect of the ischial spine, and inserts into the vaginal wall, the tendinous centre of the perineum, the anal canal and lateral border of the coccyx. Lavator ani is comprised of pubovaginalis, puborectalis, pubococcygeus and iliococcygeus (Fig. 3.8). Distention of the birth canal during

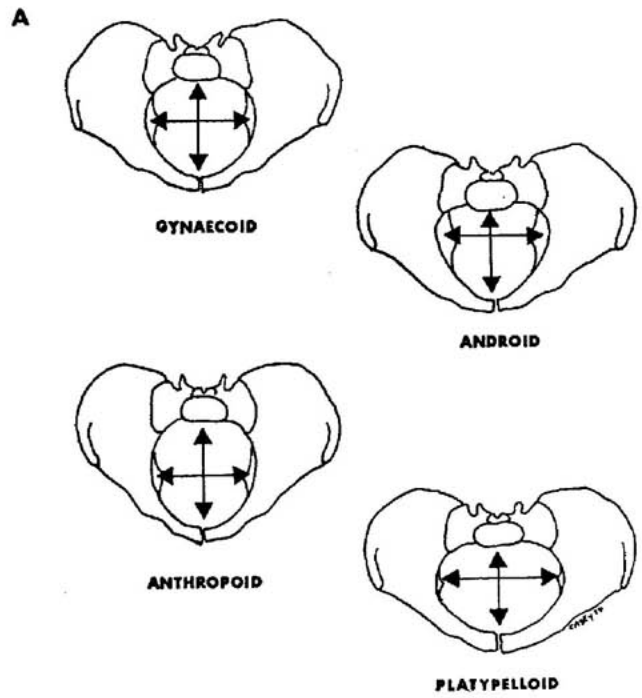

Fig. 3.7. The types of maternal plexes.
A. The shape of the brim.
B. Comparing the inlet, cavity and intertuberous diameters of different types of pelves.

the second stage of labour stretches (and sometimes tears) the muscle fibres. If marked, this damage predisposes to prolapse.

The Physiology of Labour II : The Passenger

1. **The Foetal Skull** (Figs. 3.9, 3.10).

   a. *Subdivisions* –

      i. *Base* – large, firmly united bones. They are incompressible and serve to protect the vital centres of the brain stem.

      ii. *Face* – firm, but incompletely ossified bones.

      iii. *Vault* – thin, poorly ossified bones including the occiput, two parietal bones, two temporal and two frontal bones.

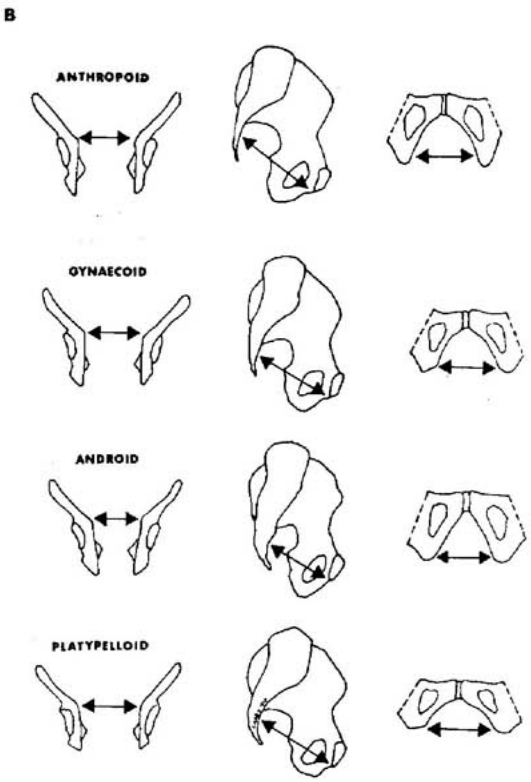

Fig. 3.7. B.

b. *Landmarks* –

i. *Anterior fontanelle* (Bregma) - diamond shape area formed by the junction of the sagittal, coronal, and frontal sutures. Measures about 3 cms. x 2 cms.

ii. *Posterior fontanelle* – a triangular shaped area formed by the junction of the sagittal and lambdoid sutures– it has only *three* sutures running into it.

iii. *The vertex* – point lying midway between anterior and posterior fontanelles.

iv. *The occiput* – from the posterior fontanelle to the foramen magnum.

v. *The sinciput* – from the bregma to the root of the nose (glabella), bounded by the coronal sutures and the orbital ridges.

vi. *The glabella* – the elevated region between the two orbital ridges.

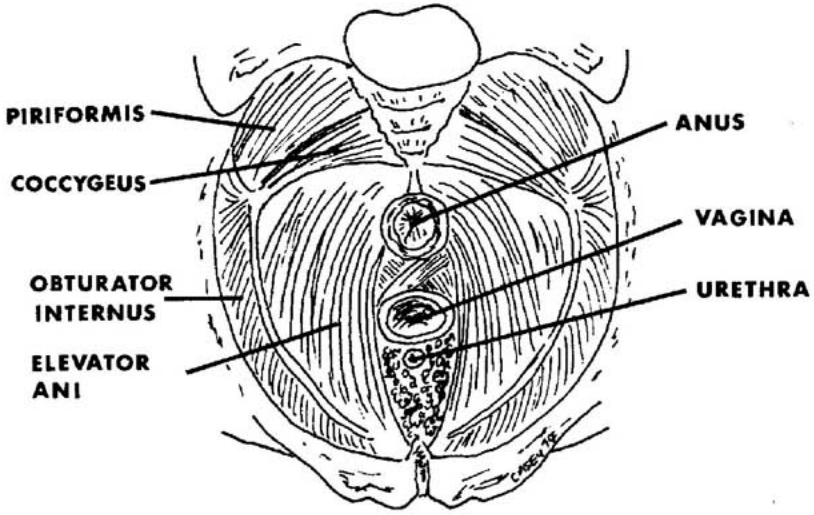

Fig. 3.8. The pelvic floor from above – note contours.

c. *Diameters* – (q.v. Table 3.3 and Figs. 3.10, 3.11).

i. *Sub-occipito-Bregmatic* – from centre of bregma to the nape of the neck.

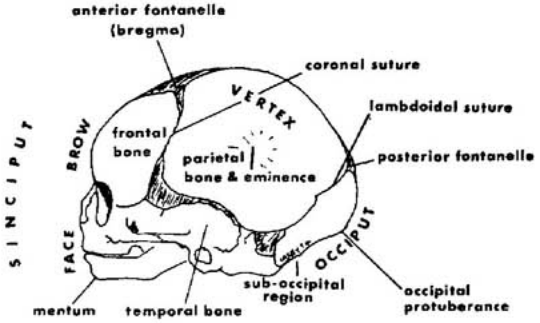

Fig. 3.9. The foetal skull – showing landmarks (side view).

ii. *Occipito frontal* – from glabella to the occipital protruberance.

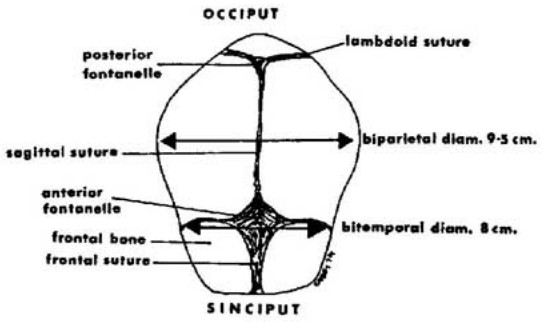

Fig. 3.10. The foetal skull (from above) showing landmarks and some diameters.

iii. *Mentovertical* – from the of chin to 1" in front of the posterior fontanella.

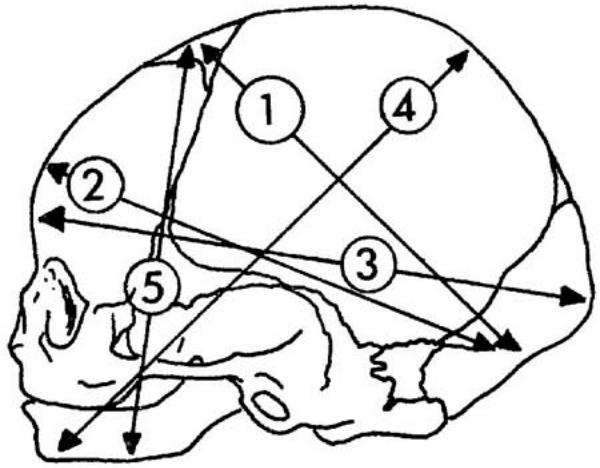

Fig. 3.11. Major diameters of the foetal skull

Diameter	Length	Presentation
1. Suboccipito-bregmatic	9.5 cm.	Flexed vertex
2. Suboccipito-frontal	10.5 cm.	Partially deflexed vertex
3. Occipito-frontal	11.5 cm.	Deflexed vertex
4. Mento-vertical	13.5 cm.	Brow
5. Submento-bregmatic	9.5 cm.	Face

iv. *Sub-mento-Bregmatic* – from the junction of neck and chin to the centre of the bregma.

v. *Biparietal* – between the two parietal eminences (the widest transverse diameter).

2. Foeto-pelvic relationships

a. *Lie* – the relationship of the long axis of the foetus to the long axis of the mother. These axes are usually parallel, i.e. longitudinal lie.

b. *Presentation* – the part of the foetus occupying the lower pole of the uterus – hence "presents". Normally it is *vertex* presentation.

c. *Position* – relationship of the denominator (a particular part of the presenting part) to the pelvic brim. In a normal flexed vertex presentation the occiput is the denominator. At the onset of labour the most frequent position is the lateral (Fig. 3.12).

d. *Attitude* – the relative position of foetal parts to one another. Normally there is universal flexion.

e. *Engagement* – when the biparietal diameter of the foetal skull enters the pelvic brim.

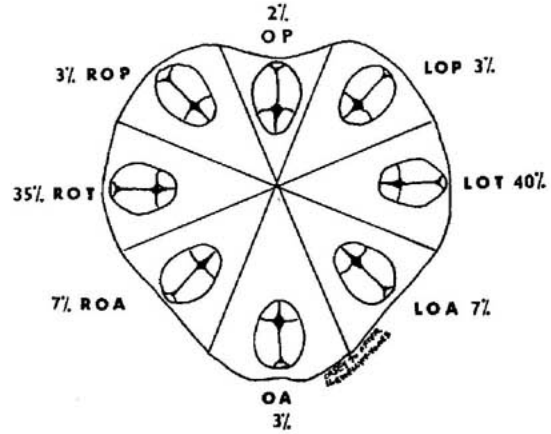

Fig. 3.12. Frequency of positions of the occiput at the onset of labour.

3. **Asynclitism** – refers to the situation where the parietal emminence presents at the pelvic inlet at the onset or labour.

   Posterior asynclitism occurs particularly in the primiparae as there is better tone in the abdominal wall and the uterus is held upright. Under these conditions the posterior parietal bone is lower than the anterior one and the sagittal suture is closer to the symphysis pubis. This is the most common mechanism of engagement (Fig. 3.13-a).

   Anterior asynclitism occurs in women with lax abdominal muscles. The uterus falls forwards so that when the foetal head enters the pelvis the anterior parietal bone is lowermost and the sagittal suture lies closer to the sacral promontory (Fig. 3.13-b).

Table 3.3: **Summary of Foetal Diameters**

	Diameter	*Length (cms.)*	*Presentation*
1.	Suboccipto-Bregmatic	9.5	flexed vertex
2.	Occipito-Frontal	11.5	deflexed vertex
3.	Occipito-Mental	13.5	
4.	Mento-Vertical	13.5	brow
5.	Submento-Bregmatic	9.5	face
6.	Biparietal	9.5	
7.	Bitemporal	8.0	
8.	Bisacromial	11.0	
9.	Bitrochanteric	10.0	

The Physiology of Labour III : The Powers

1. **The Myometrium**

   a. Is derived from the Mullerian ducts.

   b. Is composed of 3 layers –

      . an interdigitating spiral layer,
      . an outer longitudinal layer, and
      . an inner circular layer
      (the two latter ones are unimportant).

   c. The *spiral layer* is more marked in the body of the uterus, where the fibres intersect at an angle of 60°, than in the lower segment where the fibres intersect at almost 180°. The spirals uncoil during pregnancy.

   d. There is a resting tone in uterine muscle of about 6-12 mmHg.

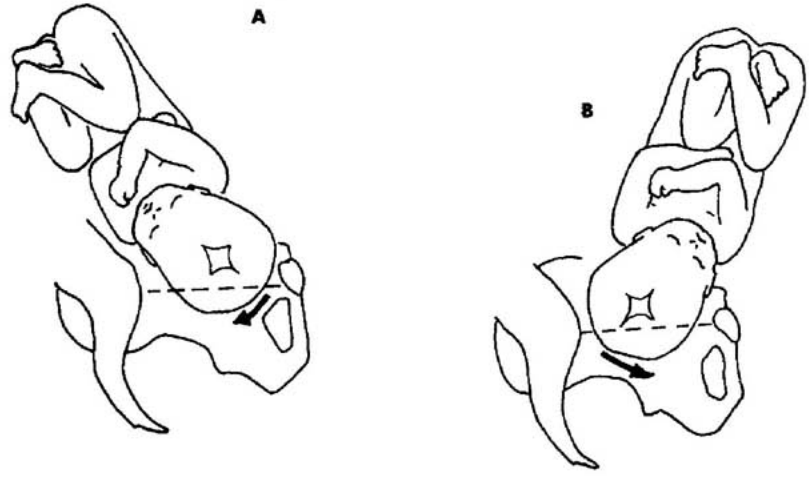

Fig. 3.13 Asynclitism:
A. Posterior asynclitism
B. Anterior asynclitism

2. **Contractions** –

   a. Originate at a pacemaker at the junction of the fallopian tube and body of the uterus.

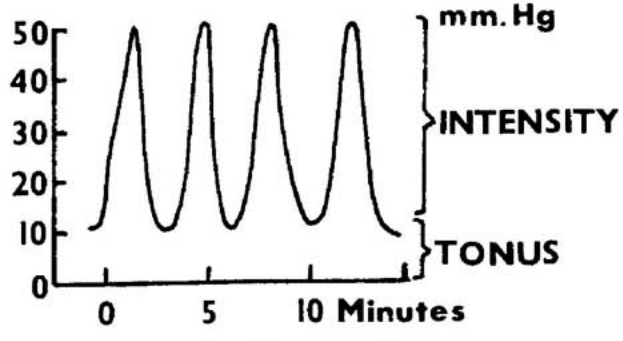

Fig. 3.14. Uterine contractions – intensity and frequency.

b. Cause an increase in intrauterine pressure (amplitude) (Fig. 3.14).

c. Produce, in the lower segment, an upward pull on the fibres of the cervix.

d. Produce circular narrowing in the upper segment and predominantly centrally directed pressure, hence the amniotic fluid flows into the lower segment causing a bulging of the forewaters with each contraction; and, direct transmission of the pressure to the foetus.

e. Cause the uterus to become more rigid and to move forwards.

f. Straighten out the foetal spine, forcing the presenting part towards the pelvis.

Mechanism of Normal Labour

The mechanism of labour refers to the way the foetus adapts to and passes through the maternal pelvis. It involves –

1. Descent
2. Flexion
3. Internal Rotation
4. Extension
5. Restitution
6. External Rotation

1. Descent

Descent is continuous throughout labour and on it the other movements are superimposed. It includes engagement which occurs often several weeks before labour in the primipara and as late as the second stage of labour in multiparous patients.

2. Flexion

At the commencement of labour the foetus is normally in an attitude of flexion. With descent, and resistence encountered at the pelvic inlet or pelvic floor there is increased flexion – the chin approaches the chest and the occiput of the foetal skull becomes lowermost (Fig. 3.15).

The increased flexion results in the engagement of the smaller suboccipito-bregmatic diameter (9.5 cms.) rather than the occipito-frontal diameter (11·5 cm.).

3. Internal Rotation

As descent through the birth canal proceeds, the foetal head rotates so that its long axis occupies the largest diameters of the pelvis. Hence, the anteroposterior diameter of the head must occupy the transverse (or oblique) diameter of the pelvic inlet and the antero-posterior diameter of the outlet. This occurs with anterior rotation of the occiput during descent – the head performing a spiral movement.

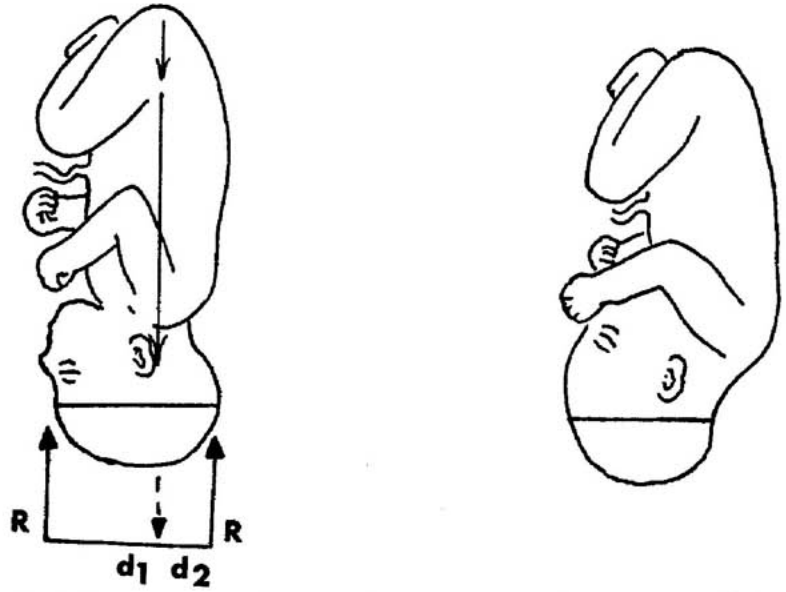

Fig. 3.15. Flexion – Resistance at the pelvic brim and the asymmetry of the foetal head encourage flexion of the neck.
d1 and d2 = distances to foreamen magnum.
R = Resistance at pelvic brim.
R x d1 is greater than R x d2. This results in flexion.

The shoulders do not rotate with the head. When the occiput is anterior the shoulders remain in the oblique diameter of the pelvic brim – the neck thus being twisted at 45° (Fig. 3.16). This relationship continues as long as the head is in the pelvis. The cause of anterior rotation is unknown. Theories suggested include –

a. *Hart's Law:*

"The part of the foetus which first encounters the resistence of

the lateral portion of the posterior segment of the pelvic floor will be rotated to the front." This depends on the fact that the pelvic floor slopes forwards, downwards and inwards, especially in its posterior part.
In most cases the occiput is first to come in contact with the pelvic floor as the head is usually well flexed.

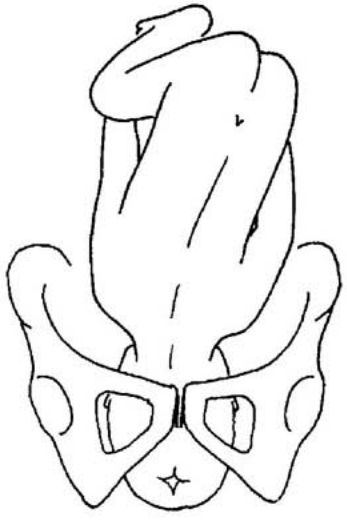

Fig. 3.16. Internal rotation of the occiput produces a 45° twist in the neck.

b. *Law of Unequal Flexibilities:*

The strongest bands of muscle connecting the head and the trunk will occupy the position where they are subjected to the least strain. The posterior muscles of the neck come to lie anteriorly – the weaker muscles of anterior part of the neck then lie in the curved posterior part of the birth canal.

c. *The Properties of the Birth Canal:*
Factors such as the bony pelvis, the elastic properties of the walls of the birth canal and the oscillatory movements produced by the uterine contractions may combine to encourage anterior rotation of the occiput.

d. *The Shape of the Foetal Head* may also be important. The head is asymmetrical with the greater proportion lying

anterior to the mento-vertical diameter (i.e. longest diameter of the foetal skull), (Fig. 3.17).

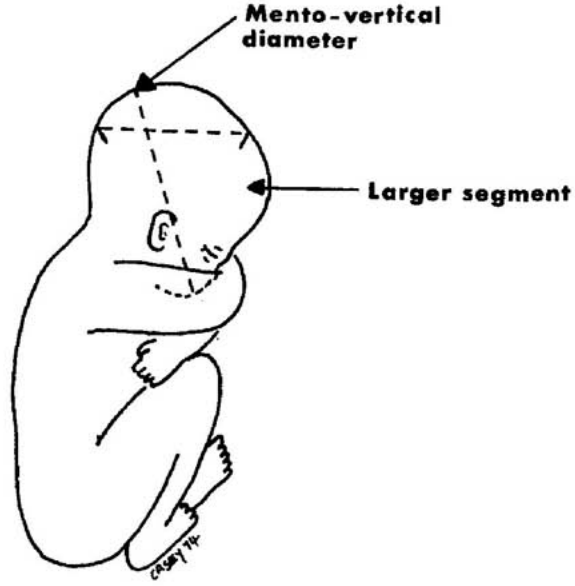

Fig. 3.17. The major part of the foetal head lies anterior to the mento-vertical diameter.

At the level of the ischial spines the birth canal curves forward providing a great deal more room posteriorly (in the hollow of the sacrum) than anteriorly. Thus, the larger segment of the foetal head is encouraged to rotate to the back.

4. **Extension**

In birth of the head the sinciput must travel much further than the occiput. This occurs with the occiput pivoting around the symphysis pubis and the head extending – the forehead, nose, mouth and chin being born in succession. At this stage, the shoulders are in the oblique diameter of the mid pelvis.

5. **Restitution**

During internal rotation the neck became twisted on the shoulders. When the head is born it is no longer restrained by the birth canal and it rotates (restitutes) back 45° – the normal head-to-shoulder relationship.

6. **External Rotation**

External rotation of the head merely reflects the internal rotation of the shoulders. After the shoulders enter the pelvis, in either the oblique or transverse diameter, they rotate back to the antero-posterior diameter – the largest diameter of the outlet. As the head is not restricted by the birth canal it rotates freely with the shoulders.

Moulding:

During labour the bones of the vault over-ride one another, thus reducing the diameter engaging in the pelvis. Moulding refers to this ability to adapt to the maternal pelvis. The engaging diameter is reduced and the diameter perpendicular to it is increased in length. The occipital bone and the two frontal bones are driven slightly under the parietal bones. The posterior parietal bone is driven under the anterior one.

Mechanism of the Shoulders, Trunk and Extremities:

The anterior shoulder reaches the pelvic floor first and rotates anteriorly – that is, in the opposite direction to the anterior rotation of the occiput. The anterior shoulder is born by lateral flexion (posteriorly), (Fig. 3.18). The posterior shoulder follows – again by lateral flexion, but in an anterior direction (Fig. 3.19). Once the shoulders are born the trunk and extremities follow with no special mechanism.

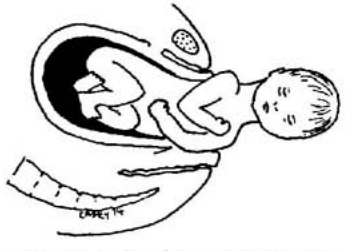

Fig 3.18. The anterior shoulder is born by lateral flexion, in a posterior direction.

Mechanism of the Third Stage of Labour:

The placenta separates from the uterine wall when retraction has reduced the placental site by about 5°%. Separation occurs through the compact decidua basalis – the myometrium acting as a barrier on one side and Nitabuch's layer on the other protects the placenta from fragmenting irregularly.

Haemorrhage is controlled by compression of the thin walled uterine vessels by strongly retracting muscle fibres arranged in a figure-of-eight fashion. Anything which interferes with muscle retraction, e.g. incomplete placental separation, or retained placental fragments, will result in haemorrhage.

Fig. 3.19. The posterior shoulder is born by lateral flexion, in an anterior direction

The signs of placental separation have already been discussed on page 3.4.

The Management of Normal Labour

On Admission to Labour Ward

1. *The History* is recorded – this comprises the antenatal record (q.v. Chapter 1) and the time of onset of contractions, the frequency and strength of contractions, and whether the membranes have ruptured.
2. *Examination* – temperature, pulse, blood pressure.
   - The character and frequency of the uterine contractions are observed.
   - Abdominal palpation – q.v. Chapter 1, and note is made of the level of the anterior shoulder. The foetal heart rate is recorded. The degree of cervical dilatation is noted.
   - A mid-stream urine specimen is tested for pH, protein, reducing sugar and acetone.
3. A pubic toilet and enema are given.
4. The patient has a shower.

Management of the First Stage of Labour

1. *Ambulation* – is allowed early, if the membranes are intact.

Once the membranes rupture, the patient should stay in bed in order to minimise the risk of cord prolapse – unless the head is well engaged.

2. *Observations* – If labour is progressing spontaneously and there is no indication of foetal distress, such as meconium stained liquor or a slowing of the foetal heart rate, observations are carried out every 2 hours, noting –

   i. maternal pulse rate, blood pressure and temperature,

   ii. frequency, duration and strength of contractions,

   iii. abdominal palpation especially regarding engagement of the head, level of the anterior shoulder and foetal heart rate.

   Note: a. The foetal heart rate is auscultated every 15 minutes if the liquor is meconium stained or if oxytocin is used (q.v. Chapter 8).

   b. Even more frequent recording of foetal heart rate is indicated if –

   i. the liquor is stained with *fresh* meconium (i.e. a light yellow-green colour), or

   ii. any slowing or irregularity of rate is detected.

3. The patient is encouraged to pass urine every couple of hours. The urine is tested for pH, protein, glucose and ketones.

4. **Cervical dilatation** – Once the cervix is half dilated, further progress is more rapid and is associated with increased severity of pain. Determination of the extent of cervical dilatation gives an accurate indication of progress of labour. Vaginal examination is more accurate and gives more detailed information than rectal examination. It must be carried out with full aseptic technique – it does not increase the risk of infection (as compared with rectal examination) when this precaution is taken.

   Vaginal examination yields information regarding –

   i. *The cervix*

   . The condition of the cervix – soft or hard.
   . The degree of effacement and dilatation.

   ii. *The presentation*

   . Cephalic (or otherwise)
   . The presence of a caput succedaneum.
   . The station of the presenting part. The level of the ischial spines is station zero and variation from it is measured in centimetres. Above the ischial spines the station is minus 1 to minus 5 (level of the inlet) and below the spines – plus 1 to 5.

   iii. *The position*

   . The direction of sagittal suture – in the antero- posterior, oblique or tansverse diameter of the pelvis.

   . The position of the anterior or posterior fontanelle.

   . If the sagittal suture cannot be palpated – the antero-posterior diameter of the head can be determined by identifying the ear. The pinna points to the occiput.

5. **Analgesics** (q.v. Chapter 12) – are usually required by the latter part of the first stage, i.e. after half dilatation of the cervix. Analgesics most frequently used are –

   a. *Pethidine* 50 mg. IM + Sparine 25 mg. – may be repeated in 3-5 hours. In some patients a more potent analgesic is required and morphine 16 mg. is sometimes used. If the baby is born within 5 hours of the injection, Lethidrone, 0.5 mg. is injected into the umbilical vein immediately after delivery and before the cord stops pulsating.

   b. *Inhalational analgesia* – 70% nitrous oxide and 30% oxygen. The patient should be instructed how to use the mask –

   i. When she feels a contraction beginning she should breathe through the mask (firmly applied to the face.)

   ii. She should not wait till the contraction becomes painful.

   iii. If she starts to lose consciousness, she will allow the mask to fall away from her face.

   iv. Between contractions she should breathe air.

   c. *Continuous epidural anaesthesia*

6. **Nutrition**

   a. In *early* first stage, where inhalation anaesthesia is not to be used at delivery the patient may have a light meal.

   b. Fluids may be taken by mouth.

   c. At a *later* stage nutrition and hydration are often achieved by intravenous infusion of 5 % dextrose. Other advantages of having a drip set up include –

      i. A route for rapid administration of anaesthetics, blood or plasma expanders (if necessary during the third or fourth stage).

      ii. Route to administer syntocinon if uterine action is inadequate.

7. **Reassurance**

   Empathy, support and encouragement for the patient are important. Relaxation accelerates labour. Bearing down too early delays cervical dilatation (the cervix becomes large and oedematous), the patient becomes exhausted and it predisposes to prolapse at a later date.
   In addition there is only a limited amount of analgesics that can be given to a patient – although the introduction of a continuous epidural anaesthetic has markedly reduced this difficulty.

Management of the Second Stage of Labour:

1. **Aseptic Technique**

   a. *Washing technique*

   A brush may be used to clean under and around the nails. If washing with soap, the hands and arms should be thoroughly washed several times with the soap and the excess lather washed off several times. The whole process should take not less than five minutes. A scrubbing brush should *not* be used on hands and arms as it tends to traumatise the skin and increase the surface bacterial count.

   If washing with a surface antiseptic preparation then two minutes should elapse during which time the preparation in use remains in contact with the skin and is *not* washed off till the end of the 'scrub-up'

   b. *Gowns and gloves* must be used from sterile packs. An efficient mask will reduce the droplet spread of infection, but when worn for much more than half an hour it becomes inefficient.

2. **Delivery Position**

   Patients will be delivered in the dorsal or lithotomy position. The lithotomy position is the method of choice for it enables easier access to the perineal area by one or more accoucheurs. There is more space to allow delivery of the head, particularly when manipulation is required to deliver the anterior shoulder. Finally, because all liquor and meconium drop immediately into the linen tub there is no risk that the foetus will inhale these fluids as the head is pushed backwards to deliver the anterior shoulder.
   If using the dorsal position a small pillow should be placed under the buttocks to elevate and make delivery easier.
   If in the lithotomy position, the buttocks should come down to the end of the bed.

3. **Swabbing**

   Using *sponge-holding forceps* the mons pubis and inner sides of both thighs, for a distance of six inches away from the vagina, are swabbed with an antiseptic solution, making sure the swab passes from in front backwards and is discarded on reaching the perianal region.

   Next the labia are separated and the vaginal introitus, periurethral and fourchette areas are swabbed.

4. **Draping**

   If in the *lithotomy position* the patient is draped by passing a large towel over each leg from inside the thigh. One side of the drape hangs down over the leg and inner thigh whilst the other side hangs down outside the lithotomy pole. The drape which is folded over near the foot is doubled back and clipped behind the lithotomy pole in envelope position. After both legs are covered, a third large towel is laid on the abdomen and the lower edge adjusted to come down to the mons pubis. Finally a perineal towel is placed in position.

   The patient is now ready for delivery.
   Draping in the *dorsal position.* A towel is laid over each flexed thigh to cover the exposed areas leaving the vulva uncovered. A third towel is placed over the abdomen and the perineal towel

placed in position. A fourth towel is placed on the bed between the thighs and the upper edge pushed under the buttocks.

5. **Catheterisation**

If there is any indication that the bladder has not been emptied it will be necessary to catheterise the patient. Chlorhexidine (hibitane) cream should be used to cover the urethral orifice and the catheter passed into the bladder through a layer of this cream.

There is 10% risk of introducing infection into the urinary tract during this procedure.

6. **Analgesia**

There are many ways of achieving adequate analgesia during delivery. These include–

a. Pudendal block – either transvaginal or transperineal.

b. Epidural anaesthesia – either continuous or single shot.

c. Oxygen and nitrous oxide (inhalation).
(Refer to Chapter 12).

7. **Episiotomy**

In up to 50% of primiparae the perineum either delays birth of the head or tends to tear rather than stretch. In these cases, it is preferable to carry out an episiotomy.

8. **Delivery**

The accoucheur may stand on whichever side he finds convenient, but most people will stand on the patient's right side.
With the right hand covered by a towel to protect the gloved hand from contamination by the anus, an attempt is made to "catch" the foetal chin as the head is being born. The chin can usually first be palpated and held about the region of the anus and is then eased across the perineum. The patient is told to stop bearing down at this stage.

Whilst the right hand is being used to ease the head out of the vaginal introitus, the left hand is used to steady the head as it "crowns" and is delivered under the symphysis pubis. This steadying of the head prevents too rapid a delivery and consequent damage to foetal and maternal tissue. Excessive guarding of the perineum, however, may delay delivery, stretch the pubo-cervical

fascia and predispose to cystocele and stress incontinence at a later date.

Once the brow and face are delivered across the perineum the towel is dropped and the index finger run round the introitus to make certain that the mouth and chin are free.

Restitution and external rotation will now take place (with the occiput turning to the left in a baby who had been in the L.O.A. position).

At this stage when using the lithotomy position it is often more convenient for the accoucheur to readjust his stance. He should take up a position so that he faces the foetal occiput. By so doing, the foetal mouth and nose are away from accoucheur and are convenient for naso-pharyngeal aspiration by the assistant.

With the right hand over the right parietal bone of the infant the head is pressed downwards to ascertain if there is any cord around the neck. If a loop of cord is seen this may be pulled down and slipped over the foetal skull.

If there are several loops of cord use two artery forceps to clamp and then cut the cord. When it is certain the neck is free of cord. the anterior shoulder is delivered.

In the L.O.A. position the right hand is placed on the right side of the baby's skull with maximum effort over the parietal bone, and the left hand over the left parietal bone. Making sure that the finger tips are not around the neck of the baby traction is exerted downwards and away from the vagina. The anterior shoulder will now slip from under the symphysis pubis. Placing the right middle finger in the axilla from behind the head is now lifted upwards and away so that the posterior shoulder is freed.

With middle finger in each axilla from behind the baby is delivered across the accoucheur's body till the legs are visible.

With the left hand supporting the shoulders and head the right hand is passed along the back of the baby to the legs and the ankles are grasped with the index finger between the ankles.

The baby is then held along the left arm in a head down position with the mouth directed towards the sister for naso-pharyngeal suction.

When the accoucheur is satisfied that the airway is clear the baby

is placed diagonally on the mother's lower abdomen with the head directed towards the mother's head.

The cord is then clamped (at about 3 cms. from the umbilicus), divided, and the baby passed to the care of the sister. 1 to 2½ cms. should be left beyond the clamp.

9. **Naso-pharyngeal suction** – to remove mucus, liquor or meconium

   a. Wipe away secretions from mouth.

   b. Suck out pharynx – remove secretions or these will be inhaled.

   c. Nasal suction – remember that the nasal cavity projects directly backwards (not upwards).

   Note: The suction tube is not an instrument for stimulation but only for the removal of secretions.

Management of the Third Stage of Labour:

1. a. In Rh positive women oxytocin (5 units) is injected intramuscularly with the delivery of the anterior shoulder of the baby.

   b. In Rh negative women the injection is delayed until the clamp is taken off the segment of cord attached to the placenta in order to reduce the possibility of foetal Rh positive cells reaching the maternal circulation.

   Note: The blood in the placenta is foetal blood and if the pressure builds up at the placental site escape of foetal cells may be facilitated.

2. **Separation of the placenta**

   This often occurs within 5 minutes of delivery of the infant but may take as long as 15-30 minutes. The signs of separation have been described (see page 56).

3. **Delivery of the placenta**

   When the uterus is felt to be firmly contracted the placenta can be delivered by the Brandt-Andrews technique. This involves –

   a. stabilising the uterus by pressure through the anterior abdominal wall,
   and

   b. traction on the cord.

   If the uterus is not firmly contracted before traction is exerted there is the risk of causing inversion of the uterus (q.v. Chapter 9).

4. **Following delivery** of the placenta ergometrine (0.5 mg. intramuscularly) is given *provided* the blood pressure is not elevated (greater than 140/90 mmHg.)

5. **Inspection of the placenta** to ensure –

   a. the placenta is complete,
   b. membranes are complete,
   c. there are three cord vessels,
   d. no placental vessels extend past the edge – suggesting a succenturiate lobe which may have remained in the uterus,
   e. weight.

6. **Repair of Episiotomy**

   a. *Vaginal wall* – with 00 catgut, first suture above apex of the incision, carefully ensuring anatomical apposition down as far as the remnants of the hymen.

   b. *Perineum* – deep sutures to obliterate dead space,
   – subcuticular suture to approximate the skin.

   Note: If pudendal block was carried out prior to delivery this should provide sufficient analgesia.

Management of the Fourth Stage of Labour

The patient is observed carefully in the delivery room for an hour following delivery – blood pressure, pulse rate and signs of haemorrhage being checked.

The uterus should be palpated abdominally to see that it is firm, not "boggy" due to retained placental fragments, and haemorrhage.

Care and Assessment of the Neonate

Following naso-pharyngeal suction the baby achieves spontaneous sustained respiration within a minute. Konakion 1 mg. is injected intramuscularly. Before being placed in the cot the baby is given to the mother to hold.

If the baby is slow to breathe it may be stimulated by slapping *on*

the sole of the foot. Those infants who have not cried within one minute require further treatment.

The vital signs are assessed at 1 minute and 5 minutes and a score given out of 10, by the Apgar system (Table 3.4). The most important signs are heart rate and respiratory effort. A score of 7-10 indicates no depression; of 4-6 some depression, and of less than 4 severe depression.

Table 3.4:

Apgar Scoring method for Evaluating the Infant

Sign	0	1	2
Colour	Blue; pale	Body pink; extremities blue	Completely pink
Respiratory effort	Absent	Weak cry; hypoventi-lation	Good, strong cry
Muscle tone	Limp	Some flexion of extremities	Active motion; extremities well flexed
Reflex irritability (response to stimulation of sole of foot	No response	Grimace	Cry
Heart rate	Absent	Slow (below 100)	Fast (over 100)

Before the baby is removed from the delivery room its surname is painted on its chest with gentian violet and later a name band is placed around its wrist.

The baby is washed and examined in a heated room as it has poor temperature regulation. The umbilical stump is painted with iodine. Examination includes –

1. Assessment of gestational age.
2. Noting any congenital defect especially –

i. midline spinal defects,
ii. hair lip or cleft palate,
iii. obvious cataracts,
iv. congenital dislocation of the hips,
v. undescended testes,
vi. imperforate anus,
vii. deformities of the extremities.

3. Recording weight, length and head circumstance.
4. Any marks or deformities received during delivery, e.g. caput succedaneum or cephalhaematoma.
These must be explained to the mother who is reassured that they will disappear.

The baby is then dressed, briefly given to the mother again, before being replaced in the heated room.

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CHAPTER 4

DISPROPORTION, PROLONGED LABOUR, OCCIPITO-POSTERIOR AND TRIAL OF LABOUR

General Instructional Objective

Recognises dystocia and its causes and implications so that management can be indicated.

Specific Behaviours

1. Distinguishes between normal labour and dystocia by history and observation.
2. Describes the causes of dystocia.
3. Describes consequences of untreated dystocia.
4. Describes the management of complications of dystocia.
5. Specifies the principles of management of the causes of dystocia.
6. Recognises malpositions causing dystocia.
7. Explains the causes of delay in the second stage of labour.
8. Anticipates the development of dystocia whilst participating in management.

Disproportion, Prolonged Labour, Occipito-Posterior and Trial of Labour

1. Definitions.

2. Causes of dystocia.
3. Clinical recognition of dystocia.
4. Management of dystocia.
5. Abnormal uterine activity.
6. Occipito-posterior position.
7. Sequelae of untreated dystocia.
8. Management of sequelae of dystocia.
9. Causes of delay in second stage of labour and their management.

Definitions

1. **Dystocia** – a difficult labour.

2. Other commonly used terms associated with dystocia:

   a. *Prolonged labour* – labour longer than 30 hours. (Labour lasting longer than 24 hours occurs in 2% of multigravidae and 10% of primigravidae).

   b. *Disproportion* – disparity between the size of the presenting part of the foetus and the maternal birth canal.

   c. *Cephalopelvic disproportion* – disproportion, where the presenting part is the foetal skull.

   d. *Contracted pelvis* – a pelvis in which one of the major diameters is reduced by more than 1½ cms.

   e. *Inertia* – slow dilatation of the cervix; primary inertia – abnormal uterine action from outset; secondary inertia – that following a period of normal action.

   f. *Inadequate uterine action* – decreased amplitude and frequency of uterine contractions.

   g. *Inco ordinate uterine action* – asynchronous contractions of different parts of the uterus.

   h. *Rigid cervix* – failure of the cervix to dilate in spite of normal activity of the uterus.

Causes of Dystocia

1. **Disproportion due to –**

   Foetal factors (present in up to 50% of cases):

   a. *Large foetus* (larger than 3½ Kg):

      i. hereditary,
      ii. grand multipara
      iii. diabetic or pre-diabetic mother,
      iv. post maturity.

   b. *Abnormal position of the occiput:*

      i. occipito-posterior.

   c. *Abnormal presentation* of foetus:

      i. face,
      ii. brow,
      iii. shoulder.

   d. *Malformation* of foetus:

      i. hydrocephalus ± spina bifida,
      ii. anencephaly,
      iii. swellings of the neck,
      iv. swellings of the abdomen (liver, kidney, spleen, bladder),
      v. "monsters".

   Maternal factors (the only abnormal factor in dystocia in up to 25% of cases):

   a. *Small pelvis*

   b. *Abnormal shapes* of maternal pelvis:

      i. android,
      ii. platypelloid,
      iii. congenital malformation.

   c. *Effect of disease or injury* (rare):

      i. traumatic,
      ii. infective (T.B., osteomyelitis),
      iii. metabolic (rickets, osteomalacia),
      iv. kyphosis, scoliosis,
      v. spondylolisthesis of lumbosacral joint,
      vi. congenital dislocation of hip,
      vii. neoplasms,
      viii. diseases of the hip or extremities.

2. **Prolonged labour** due to power failure (usually associated with mechanical causes of diproportion):

   a. *Inertia* due to poor uterine contractions.

b. *Rigid cervix.*

c. *Inco-ordinate uterine activity:*

i. colicky uterine action,
ii. hyperactive lower segment.
iii. constriction-ring dystocia,
iv. dystrophia – dystocia syndrome.

d. *Mechanical factors:*

i. congenital abnormalities of the uterus,
ii. previous operation, e.g. myomectomy,
iii. extensive fibrosis,
iv. multiple fibromyomata,
v. over distention (hydramniosis, multiple pregnancy).

3. **Excessive pain** may make labour difficult due to

a. decreased pain tolerance,

b. prolonged labour and disproportion.

It must be recognised that commonly more than one factor is operating to cause dystocia.

Clinical Recognition of Dystocia

1. **Before onset of labour:**

a. *History* – previous obstetric history, maternal disease or malformations.

b. *General Assessment* – height, general stature, gait. Some women have small pelves which, although adequate in shape, are so reduced in overall dimensions that delivery becomes difficult. This seems to be particularly so in women who are under 5 ft. 1 ins. In about 25% of these cases difficult labours are encountered, whereas in women taller than 5ft 4 ins. approximately only 5% experience problems.

c. *Careful abdominal palpation* – large head, lie, presentation, attitude, position.

d. *Clinical assessment of pelvis* – may be carried out at 34-36 weeks and repeated at the onset of labour if disproportion is suspected.

If placenta praevia is suspected (high free head or antepartum

haemorrhage) then a vaginal examination should *NOT* be done see Chapter 7.

In a vaginal assessment (i) the brim, (ii) the cavity, and (iii) the outlet are examined.

i. *Pelvic brim* – normally only the anterior third can be palpated and it usually feels well rounded. However, in small women the pelvic brim can easily be felt in almost its entirety and in those with android shapes it can be felt to have straight margins in the anterior section of the brim.

On examination, the finger passes along the iliopectineal line towards the sacroiliac joint. If this can be reached the size of the pelvis is small. It will vary with the length of the examining finger. An attempt can then be made to feel the sacral promontory. The diagonal conjugate is measured from the symphysis pubis to the sacral promontory and is usually 1-1.5 cms. greater than the true conjugate.

The shape of the pelvis, whether gynaecoid, anthropoid, android or platypelloid, can also be assessed at this examination.

ii. *The cavity* – is assessed by feeling –

. the sacral curve, normally palpable over the lower three pieces of the sacrum.
. the width of the sacrospinous ligament,
. the prominence of the ischial spines.

The ischial spines are usually not prominent but in some cases may project backwards into the pelvic cavity. Often in this type of case the sacrosciatic notch is reduced in size and the sacrospinous ligament is felt to be reduced below three-finger breadths in length.

iii. *The outlet* is assessed by measuring –

. the subpubic angle,
. the transverse diameter of the outlet,
. in cases where this is reduced, the posterior sagittal diameter.

The subpubic angle may be considerably reduced from the normal 85° angle.

Finally the ischial tuberosities should be at least 9.5 cms. (about four knuckles of a clenched hand) apart.

Munro-Kerr Manoeuvre: This manoeuvre is of special value in determining disproportion, for apart from assessing the pelvic size, the examiner also pushes the head into the brim with his left hand. In this manner the vaginal hand can be used to assess engagement and descent (Fig. 4.1).

It should be noted that many obstetricians do not carry out such an assessment during pregnancy as their intention is to proceed to a trial of labour. During this procedure clinical evaluation of the pelvis may be necessary. Their reason is that the moulding foetal head is the best "assessor" of the maternal pelvis.

Fig. 4.1. The Munro-kerr manoeuvre

e. *X-Ray Examination* – may be of immense value in managing a case of disproportion, but consideration of the time for X-ray examination is also important. In some cases where on clinical examination a degree of moderate to major disproportion is thought to exist, or the foetal head is thought

to be abnormal, or there is malposition, one is justified in ordering an X-ray of the foetus or pelvis between the 38th and 40th weeks.

Some clinicians prefer to leave X-raying the pelvis till early labour on the premise that only after labour has progressed can the degree of skull moulding, the attitude, the position of the head and the engagement of the head be accurately assessed in relationship to the maternal pelvis. Such a procedure, however, is stressful for a patient having moderate contractions, and thus becomes technically more complicated, often needing an assistant to help steady the patient during a standing lateral view.

Types of X-ray Examination:

i. *Standing lateral view of the pelvis*:

This view gives most information if only one-X-ray is to be taken at any time prior to or during labour and shows the true conjugate diameter, inclination of the pelvic brim, shape of the sacral curve, the general shape of the pelvic canal and the sacro-sciatic notch. During labour it will also show the moulding and engagement of the foetal skull.

ii. *Inlet of the pelvis*:

Brim or Thoms view allows one to determine accurately the shape of the brim. If an android shaped pelvic brim is suspected from clinical assessment, it may be of value to have an accurate view of the shape and measurements of the brim, but in the majority of cases a brim view of the pelvis gives very little extra information, and over-irradiates the foetal gonads. However, in a breech presentation it is of immense value and little irradiation of the gonads results.

iii. *Outlet view of the pelvis:*

This view may be of value when there is overall reduction in pelvic measurements and doubt also exists regarding the size of the sub-pubic angle. It is rarely necessary because the outlet is readily accessible clinically.

2. **During labour** dystocia may be recognised, by delay in progress, as determined by –

a. *Descent of the head* – if there has been no descent of the presenting part over a period of 2 hours.

b. *Cervical dilatation* – if there is no cervical dilatation during 2 hours, despite good contractions.

c. *Uterine contractions* – if contractions remain irregular, weak and ineffective.

Management of Dystocia

The management of dystocia involves either –

1. Trial of labour, or
2. Lower segment caesarean section.

1. **Trial of Labour**

The best assessor of cephalopelovic disproportion is the foetal head. Trial of labour is a clinical attempt to evaluate the extent of any disproportion, where there is a good prospect of vaginal delivery. This is the prime course of management in any case of suspected disproportion.

As a degree of difficulty may be expected a trial of labour should only be carried out in a fully equipped hospital where an emergency L.S.C.S. can be done if necessary.

Trial of labour may be induced or spontaneous and is usually the latter, unless there is some specific reason, e.g. post-maturity or diabetes. The steps involved in the conduct of a trial of labour are –

a. *Empty lower bowel* –

i. ensures no soft tissue obstruction to the descent of the presenting part,

ii. enables better application of head to lower uterine segment and cervix stimulating the release of oxytocin from the pituitary.

b. *Ambulation* may be allowed early if –

i. the patient is healthy; and

ii. the foetus is presenting normally and is in good condition.

c. *Regular observations* – every 2 hours, regarding –

i. the patient's general condition – pulse, blood pressure and temperature.

ii. contractions – frequency, duration and intensity.

iii. the foetus – abdominal palpation (lie, presentation, position, descent of anterior shoulder and engagement of the head), and foetal heart rate.

These observations should be carried out more frequently if required, e.g. when a syntocinon drip is operating or following artificial rupture of membranes, especially if liquor is meconium stained.

The patient is urged to pass urine every couple of hours –

i. as a full bladder impedes progress.

ii. to test for pH, proteinuria, ketones.

d. *Nil orally* –

i. except water, once labour is established,

ii. electrolytes and hydration are maintained by I.V. fluids.

e. *Rupture of membranes* –

i. note time,

ii. note colour of liquor,

iii. a vaginal examination is required if the head is not well engaged in the pelvis – to exclude the possibility of cord prolapse.

f. *Vaginal examinations* –

i. as infrequently as possible,

ii. to determine –

. effacement and dilatation of cervix,
. progressive descent of head,
. application of head to the cervix.

g. *Induction* – to augment a spontaneous labour if it is considered inefficient, or if preterm delivery is necessary –

i. Surgically – by either high or low rupture of membranes,

ii. Medically – with a syntocinon drip.

h. *Analgesia* – refer also to Chapter 12.

i. nitrous oxide,

ii. pethidine 50 mg + sparine 25 mg,

iii. continuous epidural anaesthesia – provided the cervix is more than half dilated,

iv. single-shot epidural anaesthetic.

i. *Trial of labour is abandoned if –*

i. There is no cervical dilatation during 2 hours, despite good contractions.

ii. After full dilatation, the head is not engaged within one hour, or the baby not delivered in 1½ hours.

iii. Foetal distress develops.

iv. Maternal condition deteriorates significantly.

v. No significant progress has been made after 24 hours' labour.

vi. Following rupture of membranes the contractions do not improve in quality and frequency in the next 4 hours.

Judgment of progress and the final decision to terminate a trial of labour is made in the light of the whole clinical impression gained by the obstetrician.

j. *Prognosis* of trial of labour –

. 50% deliver spontaneously,
. 30% require forceps,
. 20% require L.S.C.S.

2. **Elective L.S.C.S. at term** – is carried out –

a. when a trial of labour is contraindicated, i.e. with –

i. absolute disproportion,

ii. previous L.S.C.S. for disproportion,

iii. breech presentation when disproportion is suspected.

iv. malpresentation of foetal parts (except normal breech),

v. when the patient has other medical or obstetric complications such as severe pre-eclampsia, or diabetes,

vi. placenta praevia – (type II (post); type III; and type IV).

b. when there is less marked pelvic disproportion, plus other obstetric complications (e.g. "postmaturity", pre-eclampsia), which increases the risk to the foetus, or a long period of infertility.

Abnormal Uterine Activity

1. 90% of cases of dystocia associated with an abnormality of the powers are due to hypoactive or inco-ordinate uterine activity (Fig. 4.2). Inco-ordinate uterine activity is usually seen in primigravidae. The patient experiences colickyhypogastric pain and often marked backache. With inadequate activity slight (or no) pain is felt in the normal distribution and contractions appear less intense to palpation, also less frequent, less regular and less sustained.

Management involves:

a. Making sure there is no other cause for the dystocia.

b. General measures include care in noting occurence of dehydration and acidosis; review of abdominal and vaginal findings and ensuring the bladder and bowels are empty. Sedation and analgesia must be adequate.

c. *For inadequate uterine activity:*

i. Rupture membranes.

ii. Oxytocics.

iii. In second stage, forceps (or vacuum extractor) may be used. With these measures 95% of babies deliver.

iv. Antibiotics may be needed.

d. *For inco-ordinated uterine activity:*

i. Nil by mouth as 35% progress to L.S.C.S.

ii. Rupture membranes.

iii. Put the patient on her side – this simple measure may markedly reduce the pain of contractions.

iv. Analgesia – morphine or pethidine is often required. If the baby is delivered within five hours of administration of these narcotics, then levallorphan 0.1 mg. is given into the umbilical vein or intramuscularly. Analgesia is best

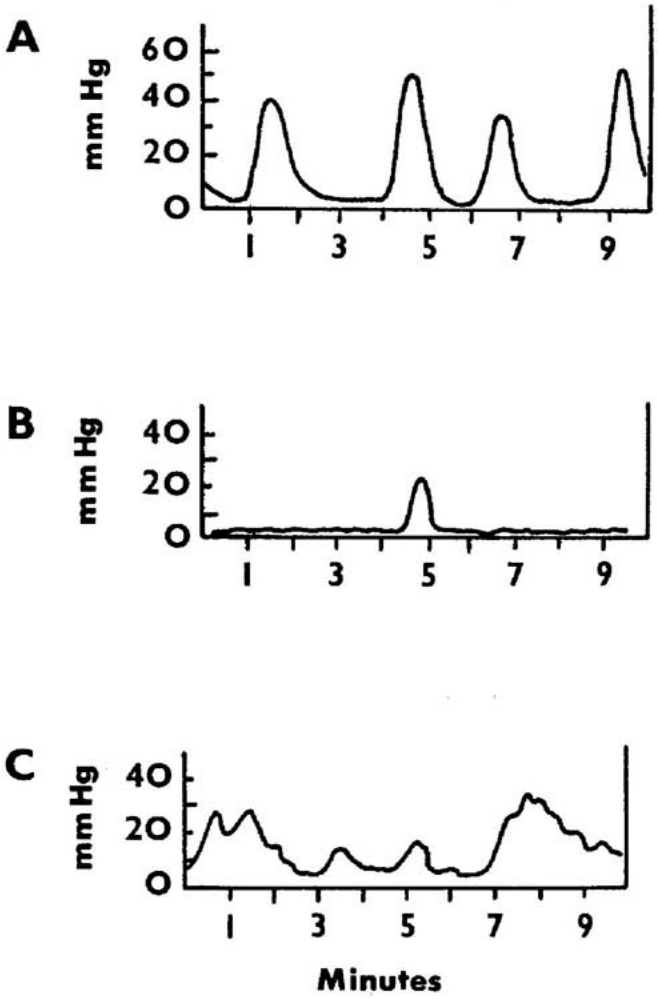

Fig. 4.2. Inadequate and incoordinate uterine activity.
A. Normal.
B. Inadequate.
C. Incoordinate.

attained by continuous epidural anaesthesia when the cervix is more than half dilated – earlier administration is likely to prolong labour and necessitate catheterisation too often.

v. Antibiotics may be required.

Note: Oxytocics augment uterine activity but do not alter the quality. Hence, unless inco-ordination is mild oxytocics are contraindicated. In mild cases some cervical dilatation can be achieved by their use.

e. With full dilatation of the cervix (and the patient in the lithotomy position) forceps delivery may be achieved if the following pre-requisites are met –

F — *full* dilatation.
O — *occipito* anterior position (except for Kielland forceps rotation)
R — *ruptured* membranes.
C — good *contractions*
E — *empty* bladder (bowel usually already empty).
P — *pain* relief is adequate.
S — head at the level of the *spines* or below.

f. Lower segment Caesarean section may be required if the cervix fails to dilate or if foetal or maternal distress supervenes.

2. **False labour** is labour in which the patient has contractions not unlike those of true labour, but there are no other signs which indicate that the patient is actually in labour and the contractions eventually pass off. In false labour the pains are irregular, do not increase in duration and severity or become more frequent – as opposed to true labour. Again, the cervix does not become effaced and there is no descent of the presenting part.

   The patient is given analgesics and sedation. The situation is explained to her, she is reassured and may be sent home.

3. **Contriction Ring Dystocia** – see Delay in Second Stage.

4. **Colicky uterus** indicates a pattern of uterine activity characterised by frequent irregular contraction occurring all over the uterus. It may be termed uterine fibrillation and is a result of spontaneous new pacemakers. There is no effective expulsive force. The patient may experience colicky abdominal cramps, backache or lower abdominal pain depending on the site of maximal contraction.

5. **Hyperactive lower segment** involves a loss of the normal gradient of intrauterine activity. The lower segment contracts more strongly than the upper – normal "fundal dominance" is lost. The patient suffers especially from backache and contractions may be severe and distressing.

   These are forms of inco-ordinate uterine activity and should be managed as described in section 1. above.

Occipito-Posterior Position

Definition:

1. Denominator – occiput lying in the posterior segment of the maternal pelvis.
2. Presenting diameter – variable, depending on the presenting part, but usually occipito-frontal (11.5 cm.).
3. Lie, longitudinal.
4. Presentation – cephalic.

(see Fig. 4.3.)

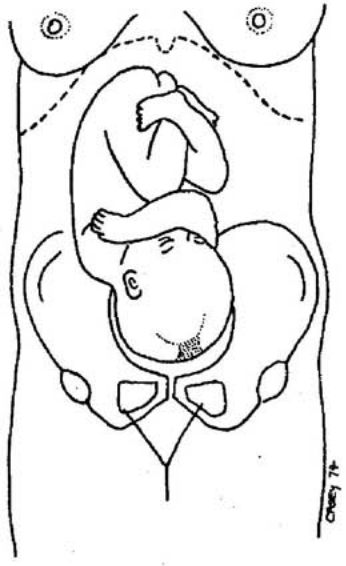

Fig. 4.3. Occiputo-posterior position.

The occiput may take up any of these positions:

1. right occipito-posterior (ROP) – where the long axis of the head is in the right oblique diameter of the pelvis,
2. left occipito-posterior (LOP) – where the long axis of the head is in the left oblique diameter of the pelvis,
3. occipito-posterior (OP) – the long axis of the head is in the antero-posterior diameter of the pelvis.

All these mal-positions are likely to cause delay in the first stage of labour.

Incidence:

There is great variation in the estimated incidence of the occipito-posterior position at the onset of labour (from 8-30%). This is due to –

1. difficulty in palpation to be sure of position,
2. majority of positions are right or left lateral, and
3. most rotate anteriorly during labour.

Related Problems:

1. Cephalopelvic disproportion – the *shape* of the inlet, especially with android or anthropoid pelves (a long AP and short transverse diameter) encourages engagement with the biparietal diameter in the transverse of the inlet.
2. When the head is deflexed at the onset of labour, a larger diameter presents at the pelvic brim.

Diagnosis:

1. **History**
   a. Occipito-posterior positions are often associate with severe backache.
   b. Prolonged infective labour.
2. **Abdominal Examination**
   a. Flattened abdominal contour (may have depression, or lack of fullness, supra-pubically or in the umbilicus).
   b. Back and shoulder well over in flank (not easily identified).

c. Limbs are felt anteriorly.

d. Often the head is high; and the brow and occiput are at the same level.

e. The finger placed at the side of the occiput sinks deeper than that over the sinciput.

f. Foetal heart sounds are loudest far out in the flank or centrally.

3. **Vaginal Examination**

a. The anterior fontanelle is in the anterior segment of the pelvis and the posterior fontanelle in the posterior segment.

b. The sagittal suture indicates the position of the occiput (as left to right).

c. If in doubt about the position, the ear may be used as a landmark – the pinna points to the occiput.

Mechanisms of Labour:

Of all occipito-posterior positions at the onset of labour –

- 80% rotate anteriorly through 135° to an occipito-anterior position,
- 10% rotate posteriorly through 45° to a persistent occipito-posterior position ("face-to-pubes"), and
- partial rotation of the occiput occurs to the lateral position with no further progress – (deep transverse arrest of the head). This usually occurs in the plane of least pelvic dimensions.

Irrespective of the rotation involved, labour will probably be prolonged due to abnormal pelvic shape, abnormal uterine activity and a large presenting diameter (due to lack of flexion and moulding). These factors combine to produce slow dilatation of the cervix, slow descent of the head and maternal distress. (Figs. 4.4 and 4.5)

1. **The Anterior Rotation**

a. *Descent and engagement* – with descent of the head through the inlet there is a tendency to deflexion resulting in engagement of the occipito-frontal diameter (11.5 cms.)

b. Internal rotation – through 135° to the occipito-anterior position is followed by normal delivery of the infant.

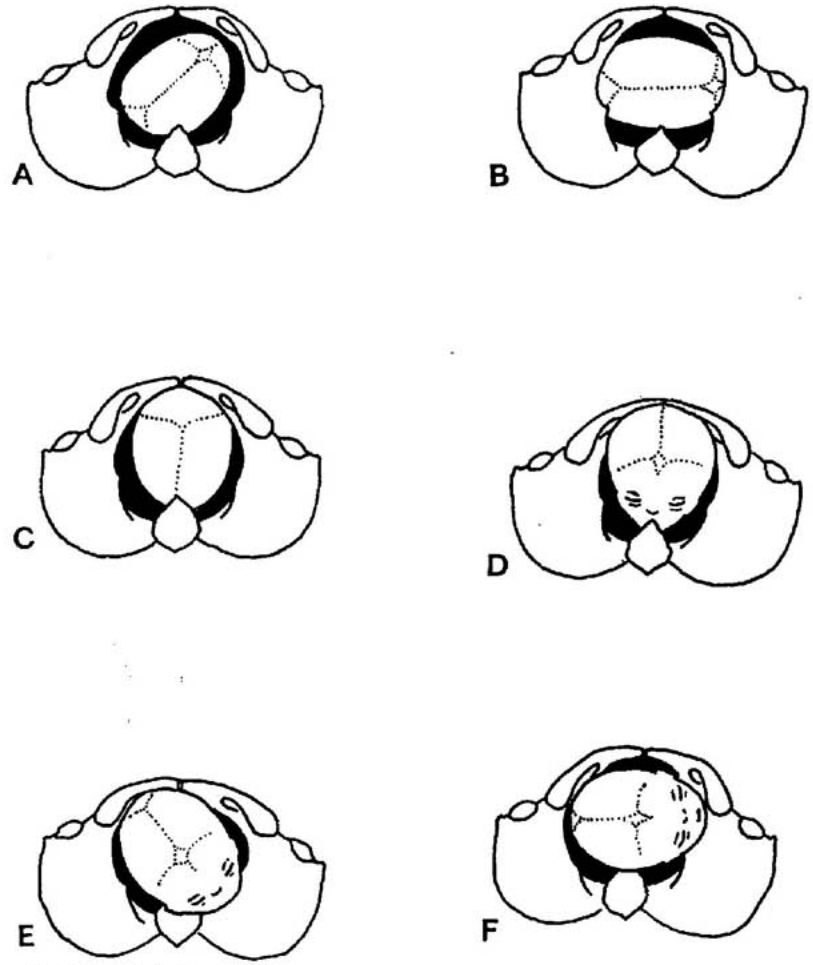

Fig. 4.4. Anterior rotation.

SUMMARY OF LONG ARC ROTATION : R.O.P. TO O.A.

A. R.O.P. onset of labour
B. Internal rotation : R.O.P. to R.O.T.
C. Internal rotation : R.O.T. to O.A.
D. Extension.
E. Restitution : O.A. to R.O.A.
F. External rotation : R.O.A. to R.O.T.

2. **The Posterior Rotation**

a. *Descent and engagement* – the head fails to flex and descends

in the oblique diameter of the pelvis, the occipito-frontal diameter (11.5 cms.) engaging.

b. *Internal rotation* – occurs through 45° so that the occiput occupies the hollow of the sacrum .The sagittal suture lies in the AP diameter of the pelvis.

c. *Delivery of the head* – occurs with further descent. There is little flexion, resulting in birth of the bregma, vertex and occiput, followed by extension – the face finally sweeping the perineum.

If there is good flexion the diameter pivoting under the symphysis pubis (the suboccipito-frontal (11.5 cms.) presents at the perineum and increases the risk of trauma to the maternal passages.

d. *Restitution* of the occiput through 45° to either the right or left oblique in order to resume the normal head and shoulder relationship.

e. *External rotation* through a further 45° brings the occiput to the lateral position as the shoulders occupy the AP diameter of the pelvis.

f. *Delivery of the trunk* follows flexion of the trunk.

3. Rotation and arrest in the transverse diameter is discussed in Section 9.

Management:

1. Prior to labour no treatment is required or possible.
2. With the onset of labour – management is trial of labour with special attention to:

   a. *General*

      i. watch for and treat dehydration and acidosis,
      ii. nil by mouth (except water),
      iii. adequate analgesia.

   b. **Vaginal examination** – to confirm position.

   c. **Oxytocin infusion** – may be used only if the contractions are weak and absolute disproportion has been excluded.

3. **Delivery**

Persistent occipito-posterior requires a generous episiotomy so that gross vaginal lacerations can be avoided.

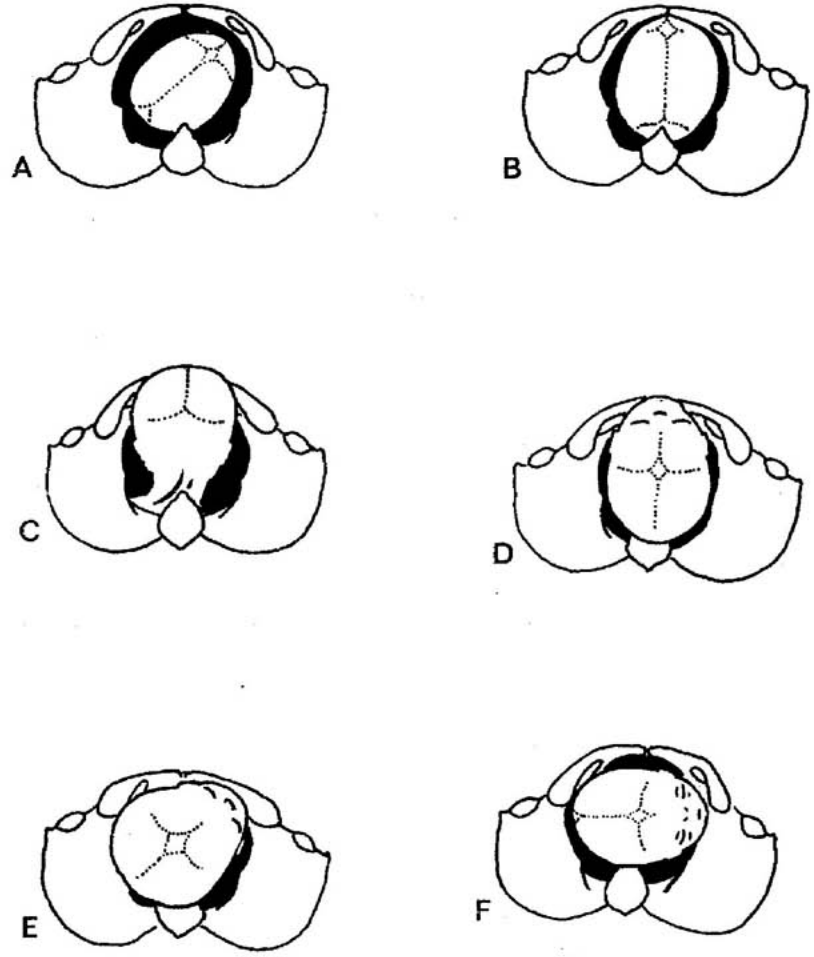

Fig. 4.5. SUMMARY OF SHORT ARC ROTATION: R.O.P. TO O.P.

A. R.O.P. : onset of labour
B. Internal rotation: R.O.P. to O.P.
C. Birth by flexion.
D. Birth by extension.
E. Restitution : O.P. to R.O.P.
F. External rotation : R.O.P. to R.O.T.

Delivery may be –

. spontaneous,
. forceps,
. L.S.C.S.

a. *Forceps delivery:*

If spontaneous delivery appears unlikely after about one and a half hours in the second stage, forceps delivery (by a specialist obstetrician) may be performed. This may involve either –

i. simple, extraction following spontaneous anterior rotation,
or
ii. rotation (either manually or with Kielland's forceps) and extraction – see management of deep transverse arrest,
or
iii. delivery as a persistent O.P only if the head is distending the perineum – i.e. as an assistance to the maternal powers. If the head is higher the incidence of trauma to mother and foetus is unacceptable.

b. *L.S.C.S.* is required if labour fails to progress or foetal distress occurs and forceps delivery is not possible. It may also be required if brow presentation develops (see Chapter 10).

Sequelae of Dystocia

1. **Foetal** – little risk until membranes rupture.
   a. Foetal death – stress asphyxia (excessive contractions cause decreased placental blood flow), greater trauma in delivery.
   b. Cerebral damage.
   c. Increased risk of infection.
   d. Increased risk of cord prolapse (increased twelve times).
   e. Cephalhaematoma.
   f. Fracture of skull.
   g. Erb's palsy – roots of C5 and C6 torn, following traumatic delivery of head and shoulders.

Increased foetal risk as often postmature.

2. **Maternal** –
   a. Increased risk of infection – prolonged labour.
   b. Increased risk of severe birth canal injury – ruptured uterus, and with assisted deliveries (especially rotations), torn cervix, vagina, traumatic fistulae.
   c. Psychological fear of further difficult pregnancy and avoidance thereof.
   d. Increased incidence of postpartum haemorrhage.
   e. Dehydration.
   f. Acidosis.
   g. Increased incidence of urinary tract infections.
   h. Trauma to bladder neck, bladder neck necrosis with formation of vesicovaginal fistula (after about 2 hours in second stage).

Management of Sequelae

1. *Foetal Death* – deliver by either –
   a. normal vaginal route,
   or
   b. destructive operation (embryotomy) –
      . craniotomy,
      . decapitation,
      . evisceration,
      . cleidotomy.
   c. L.S.C.S. – if inexperienced in the above (b.) measures.
2. *Cerebral Damage* – prophylaxis – careful forceps, judicious estimation of progress of labour.
3. **Infection** – amnionitis, due to ascending infection may produce intra-natal pneumonia in the foetus without stimulating a rise in maternal temperature. Peri-natal mortality (in both primigravidae and multigravidae) increase significantly 24 hours after the membranes rupture.

   With the exception of crystalline penicillin G given by injection in doses of 1 megaunit, the ordinary antibiotics given by mouth or intra-muscular injections do not reach the amniotic fluid in

sufficient concentration in the time available to be fully effective in controlling infection.

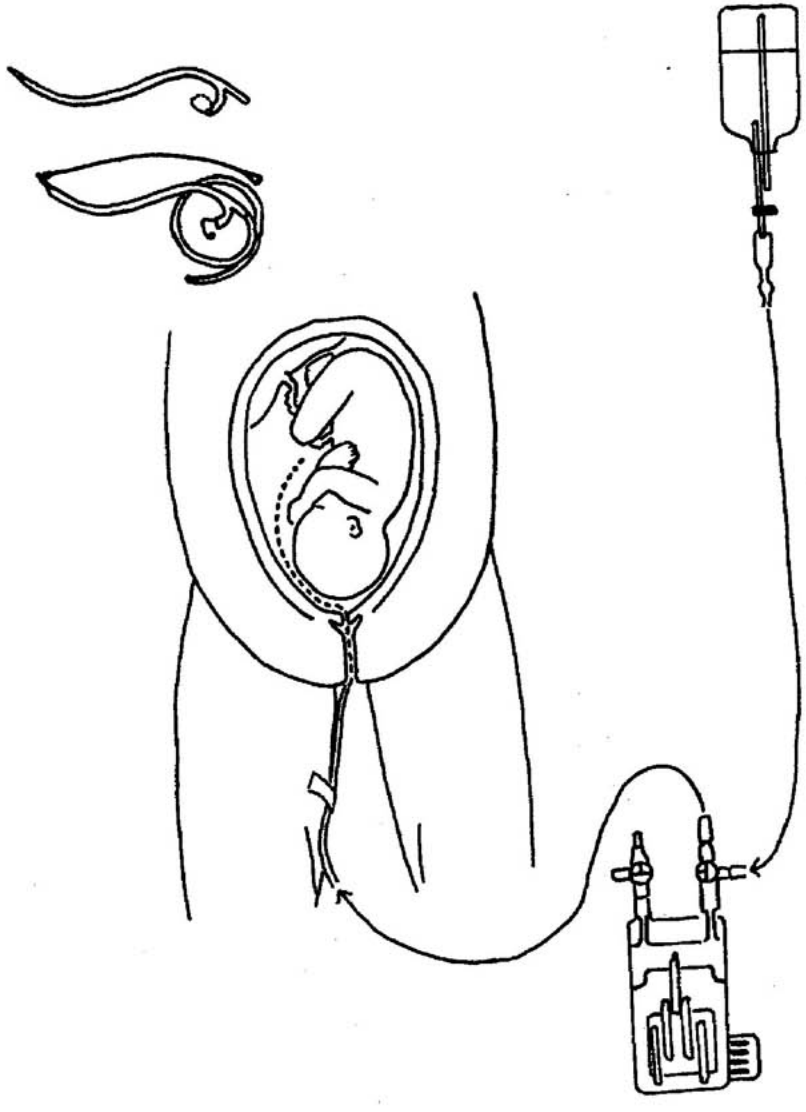

Fig. 4.6. Introduction of antibiotics into the uterine cavity using Carey's modification of the Drew-Smythe catheter.

Even with prophylactic antibiotics given orally or intra-muscularly during labour, one third of mothers who have had prolonged labour will show evidence of puerperal sepsis (see Chapter 9).

To be fully effective antibiotics must be given directly into the amniotic fluid in high concentration. A polyethylene catheter may be inserted above the presenting part, using Carey's modification of the Drew-Smythe catheter as an inserter. Streptomycin 1 gm. (or ampicillin 1 gm. is added to 500 mls. of 5% dextrose and 100 mls. of this solution is run into the amniotic cavity every two or three hours). This is the only effective way of introducing an antibiotic (except crystalline penicillin) into the amniotic fluid. (Fig. 4.6).

4. **Cord Prolapse** – requires delivery as soon as possible (unless the foetus is dead, when normal delivery is continued).
   While preparations for delivery are carried out –
   a. raise foot of bed, and
   b. get the patient into either the Sim's or genu-pectoral position – to prevent compression of the cord between the presenting part and the pelvis,
   c. administer oxygen (of doubtful value),
   d. digitally push the presenting part away from the cord – to relieve compression.

   Note: There is no risk to the mother in these procedures. If the pre-requisites for forceps delivery are fulfilled then this is the method of choice. If not, L.S.C.S. must be undertaken.

5. **Spontaneous rupture of the uterus** – usually occurs at the junction of the expanding lower segment and the retracting upper segment Fig. 4.7. It may also occur at the site of previous Caesarean section – especially if classical section had been done.

   The patient experiences severe abdominal pain and shock rapidly ensues. Vaginal blood loss is variable in amount. On examination all the signs of an acute abdomen are present and vaginally there is noted an absence of the presenting part.

   Management involves resuscitation and immediate laparotomy and hysterectomy. The baby does not survive.

   In cases where previous Caesarean sections have been carried out consideration should be given to elective L.S.C.S. at term.

After two previous Caesarean sections (L.S.C.S.), this is the method of choice for delivery. Following classical Caesarean section 2.5% of uteri rupture in a following pregnancy, but this figure rises to 10% if the mother goes into labour.

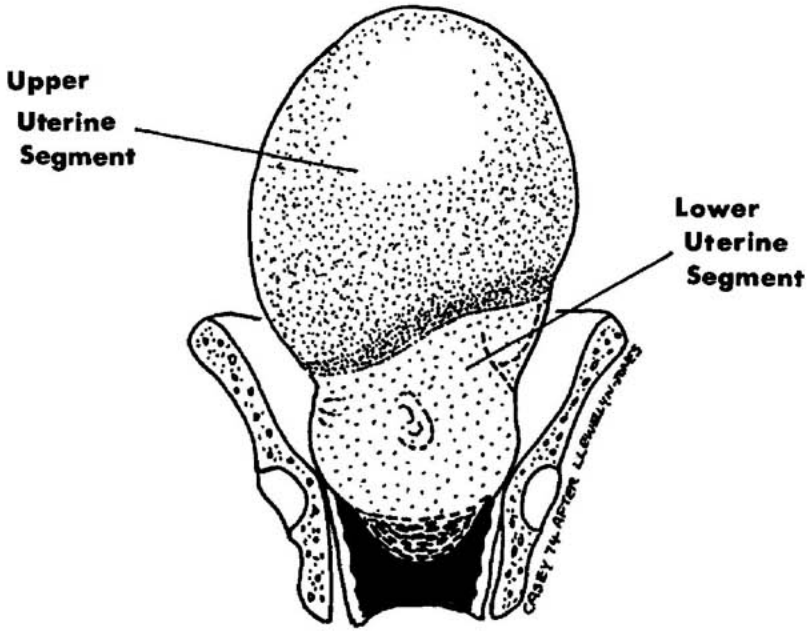

Fig. 4.7. Cephalopelvic disproportion with threatened rupture of the uterus.

6. **Dehydration and Acidosis** require the administration of 5% and 10% dextrose respectively, by intravenous drip, Acidosis can be detected by urinalysis.

   Especially in prolonged labour where operative interference may be expected, there should be no oral food given, but a dextrose drip should be running from early in the labour.

   Note: Acidosis decreases the myometrical response to oxytocin.

7. **Trauma to the bladder** –

   a. do not delay for too long in the second stage, and
   b. ensure good obstetrical manipulations.

Causes of Delay in the Second Stage of Labour

1. Deep transverse arrest.
2. Shoulder dystocia.
3. Abnormal presentation.
4. Constriction ring.
5. Rigid perineum.
6. Hypotonic uterine activity.
7. Slow normal labour.
8. Vaginal abnormality – septum.
9. Short umbilical cord.
10. Abdominal or thoracic enlargement of foetus.
11. Locked twins.

Management:

1. **Deep transverse arrest**

   A faulty condition in the mechanism of labour when a flat sacrum and android type of maternal pelvis cause arrest of the foetal head at the level of ischial spines, with the sagittal suture in the transverse diameter. The situation occurs most commonly with the funnel-shaped android pelvis.

   Successful delivery depends upon rotation of the occiput to an anterior position – such that the long axis of the head occupies the largest diameter of the pelvis. This may be achieved by –

   a. forceps,
   b. manually,
   c. the vacuum extractor.

   a. *Rotation with Kielland's forceps.*

      If immediate rotation is difficult, slight traction may be applied and rotation once again attempted. Often rotation is easier if the head is elevated slightly so that an adequate transverse diameter is obtained.

      Note: Undue force must NOT be used. If unsuccessful proceed to L.S.C.S.

   b. *Manual rotation* involves grasping the head and elevating it above the point of arrest. Rotation is accomplished with an assistant pushing the anterior shoulder across the maternal

abdomen. Delivery is completed with forceps. (Fig. 4.8).

c. Although the use of a *vacuum extractor* results in less vaginal trauma it has a high failure rate (as high as 20%) and is not a popular method for rotation.

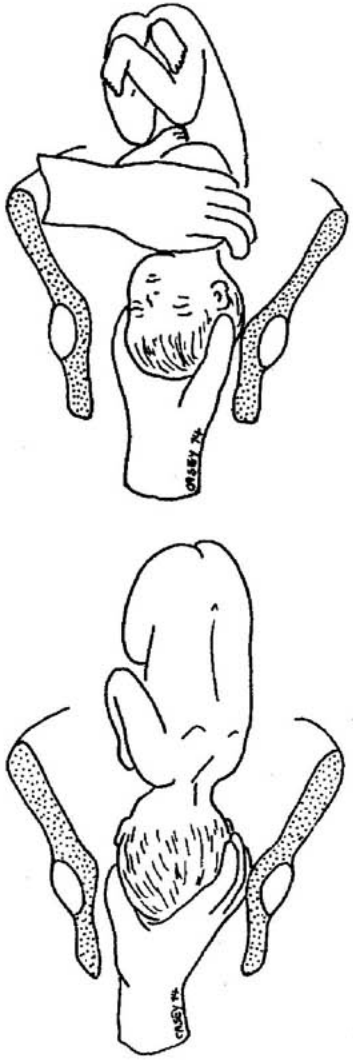

Fig. 4.8. Manual rotation in deep transverse arrest.

2. Shoulder dystocia

Following delivery of the head, impaction of the shoulders prevents further progress in an otherwise normal second stage. The baby will die if not delivered immediately. (Fig. 4.9).
Instead of rotating to negotiate the pelvic brim in the transverse or oblique diameter, the shoulders become impacted in the antero-posterior diameter.

Management involves –

a. Anaesthesia.
b. Lithotomy position.
c. Vaginal examination to exclude other causes.
d. Episiotomy.
e. *Attempt to deliver the anterior shoulder* by downward and backward traction of the baby's head, placing tension on the tissue and clavicle of the anterior shoulder, while an assistant applies firm pressure on the point of the shoulder (above the symphysis pubis) so that it may be forced down under the symphysis. (Fig. 4.10).
If this fails to work –
f. *Attempt to deliver the posterior shoulder.* The foetal head is lifted forwards and one hand passed up into the curve of the sacrum and then the fingers run up in front of the foetal shoulder to the antecubital fossa. Pressure over the flexor surface of the antecubital fossa will allow the arm to be delivered by Pinard manoeuvre down into the pelvis. By pulling on the arm the shoulder then slides down into the curve of the sacrum and pelvic cavity and delivery is usually easy. (Fig. 4.11)

If difficulty is experienced in bringing down an arm –

g. *Attempt to rotate shoulders*
   i. Several fingers are introduced into the vagina and pressure exerted behind the anterior shoulder to encourage rotation to an oblique diameter. Simultaneous suprapubic pressure is exerted by an assistant to bring the anterior shoulder into the pelvis.
   ii. Several fingers of the left hand are passed up in front of the posterior shoulder and pressure is exerted sq as to rotate it, resulting in its delivery into the pelvis. The

Fig. 4.9. Shoulder dystocia.

Fig. 4.10. Delivery of Anterior Shoulder.

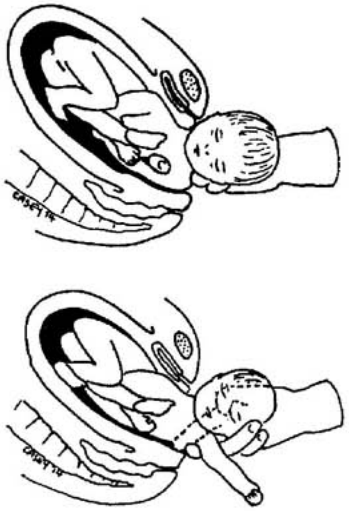

Fig. 4.11. Delivery of Posterior Shoulder.

anterior shoulder (now posterior) is delivered by repeating the procedure.

h. Finally, if no other measure has succeeded and usually when the baby is dead, the head may be pressed backwards and a cleidotomy performed using Mayo scissors.

3. **Constriction ring dystocia**

A constriction ring is a rare condition involving annular spasm of the myometrium usually at the level of the junction of the upper and lower uterine segments. Constriction may also occur–

a. in the lower uterine segment,
b. at the level of the internal os,
c. in the upper uterine segment.

It may occur spontaneously, follow the use of oxytocics in cases of hypertonic uterine action or follow obstetrical interference and is associated with extreme thinning of the lower uterine segment. Lack of recognition may lead to rupture of the uterus (in a multipara) or uterine inertia (in a primiparous patient).

Diagnosis:

a. Obstructed labour.
b. Vaginal examination reveals a poorly applied presenting part.
c. Definitive diagnosis can only be made by intra-uterine exploration when the constriction ring is palpated.
d. The constriction may be palpable, or even visible abdominally.

Treatment:

a. Surgical anaethesia to relax the ring and then subsequent forceps delivery.

b. Caesarean section when the condition has been allowed to progress for too long. It may be necessary to incise the ring (classical Caesarean section) to deliver the infant.

4. **Malpresentation** which may result in dystocia include:

. vertex – occipito-posterior position with extended head (10% of presentation),

. brow
. face – mento-posterior,

. shoulder
. breech – with larger than average head, or flexed, knee or footling breech.

Management of these condtions are considered under specific sections.

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CHAPTER 5

HYPERTENSION IN PREGNANCY

General Instructional Objective

Understands hypertension in pregnancy so that appropriate management can be instituted.

Specific Behaviours

1. Discusses aetiology of hypertension in pregnancy.
2. Describes changes associated with hypertension in pregnancy.
3. Describes the complications which may result from hypertension in pregnancy.
4. Demonstrates an ability to assess a woman with hypertension in pregnancy.
5. Discusses pharmacology of drugs influencing hypertension in pregnancy.
6. Discusses the managment of patients with hypertension in pregnancy.
7. Displays an understanding of the significance of investigations for hypertension in pregnancy.
8. Demonstrates an ability to counsel women with hypertension in pregnancy.

Hypertension in Pregnancy

Definitions:

1. **Hypertension in Pregnancy**

   A pregnant woman is said to be suffering from hypertension

when the diastolic blood pressure (B.P.) is raised 15 or more mm. Hg above pre-pregnancy levels, or when there is a sustained B.P. of 140/90 or greater.

N.B. The normal tendency is for B.P. to fall slightly during the mid trimester.

2. **Pre-Eclampsia**

A condition associated only with the pregnant state in which at least two of the following three criteria are fulfilled.

a. *Hypertension* (as defined above) – Attributable to no other cause.

b. *Oedema* – Must be generalised and attributable to no other cause.

c. *Albuminuria* – Attributable to no other cause (e.g. contamination, infection, postural proteinuria).

3. **Eclampsia**

"Fitting" for the first time, in pregnancy.

N.B. 80% of cases follow pre-eclampsia.
20% have *no* antecedent pre-eclampsia.

Hypertension In Pregnancy

Incidence:

. 6% of all pregnancies will be complicated by pre-eclampsia.
. 12% of Primigravidas will have pre-eclampsia.
When causes of hypertension other than pre-eclampsia are included the total incidence is slightly higher than that of pre-eclampsia, the incidence of cases other than those associated with pre-eclampsia increasing with maternal age.

The following conditions predispose to pre-eclampsia:

a. First pregnancy.
b. Essential Hypertension.
c. Multiple pregnancy (incidence raised three times).
d. Renal disease.
e. Hydatidiform mole (may cause pre-eclampsia in the first half of pregnancy).
f. Hydramnios.
g. *Rh*-isoimmunisation.
h. Diabetes in pregnancy.

Aetiology:

1. **Causes of hypertension in pregnancy** other than pre-eclampsia.

a. Essential hypertension.

b. Renal disorders and occlusive renal artery disease.

c. Endocrine disorders.

d. Coarctation of the aorta.

2. **Aetiology of Pre-eclampsia**

This remains unknown. Some of the theories attempting to explain it are summarised below:

a. The concept of circulating toxins producing pre-eclamptic "toxaemia" has never been validated and is now regarded as inaccurate.

b. Some have tried to relate pre-eclampsia to increased intrauterine tension using the increased incidence associated with multiple pregnancy, hydramnios, hydrops and diabetes to support their argument.

c. Dietary deficiency has been proposed as an aetiological factor but this has not been proven.

d. Dixon *et al* (1967), have revealed some interesting uterine changes occurring in pre-eclampsia by biopsying the sub-placental uterine wall in cases of pre-eclampsia which have gone to Caesarean section. They have shown that the normal vascular change (dilatation of the uterine radial arteries) does not occur to the same extent or to as great a depth in the sub-placental uterine wall of the pre-eclamptic as in non-pre-eclamptic cases. Also, an increased amount of immature trophoblast and prominent degenerative changes in other trophoblastic cells have been found, They state that the result of these changes may be a chronically reduced placental blood flow triggering the release of thrombolpastins from the placenta and thus leading to the characteristic peripheral vascular lesion of pre-eclampsia to be described below.

Pathological changes associated with hypertension in pregnancy:

With hypertension in pregnancy not due to pre-eclampsia the pathological changes are the same as those occurring in non-pregnant patients and will vary with the severity and stage of the process.

Pre-eclampsia on the other hand is associated with a characteristic lesion in the peripheral vascular bed. This lesion is thought to be due to the deposition of fibrin and in effect results in a narrowing of the vessels involved.

Increasing peripheral resistance causes a direct rise in B.P., a loss of fluid because of the increased hydrostatic pressure, and thus generalised oedema.

The peripheral vascular lesion may involve any organ of the body but most commonly and significantly affected are the placenta, kidneys, liver and brain. Involvement of the kidneys may lead to a decreased glomerular filtration rate, activation of the renin-angiotension pathway and thus a secondary increase in B.P. It may also lead to albuminuria with a consequent decrease in the osmolality of the vascular compartment and an aggravation of the oedema.

The histological appearance of the peripheral vacsular lesion is as follows.

At first there is a swelling of endothelial cells in capillaries and pre-capillary arterioles. As the condition progresses there is a fibrinoid deposition between the endothelial cells and the basement membrane. This specific lesion has been found only in pregnant patients.

Complications of Pre-eclampsia:

These will be classified according to organ involvement.

a. *Placenta*

Pre-eclampsia is associated with fibrinoid deposition on the villi and in the intervillous spaces, reducing blood flow and causing stagnation of maternal blood. Infarcts may also occur, further reducing the placental function. Normally about 500-700 ml. of maternal blood pass through the placental "lake" each minute. This may be seriously reduced in pre-eclampsia so that only 300-400 ml. pass each minute. The result is reduced foetal nutrition, failure of intrauterine growth and poor reserves of glycogen for the neonate.

Also, premature separation of the placenta is more common in patients with pre-eclampsia, the incidence of accidental haemorrhage being 10-15%.

b. *Kidneys*

Reduction in glomerular filtration rate with a secondary aggravation of B.P. rise has already been mentioned.
Albuminuria in pre-eclampsia is due to a change in permeability of the glomerular capillaries. It is an unfavourable prognostic sign being associated with an increased foetal wastage and a worsened maternal prognosis.

c. *Liver*

Periportal destruction and necrosis occur due to fibrinoid deposition within the portal vessels. There is also occasional haemorrhage within the liver but regeneration usually occurs over some days without serious residual damage. Occasionally liver rupture occurs.

Rarely, liver involvment may lead to disorders in the clotting mechanism with a consequent haemorrhagic diathesis.

d. *Lungs*

In advanced pre-eclampsia the lungs may show features of acute pulmonary oedema. This may be a part of the generalised oedema of the condition or may be secondary to heart failure.

Hypostatic pneumonia may also occur especially in those patients bedridden for considerable lengths of time.

e. *Heart*

Microvascular cardiac lesions do occur but are considered to be an inadequate explanation of the acute congestive cardiac failure which may cause maternal death in pre-eclampsia. It is more likely that cardiac failure is due to increased resistance in the peripheral circulation, a reduced intra-vascular compartment and decreased cardiac return.

f. *Brain*

Post mortem findings in patients who die following eclampsia vary from focal or extensive petechial haemorrhages to gross cerebral haemorrhage, and they may occur in the cerebral cortex, in the parabasal ganglia, and occasionally in the pons.
These lesions are probably the result of severe elevation of blood pressure and may contribute to the production of eclampsia. The eclampsia alternatively may be caused by other factors and

may lead to cerebral haemorrhage via the production of cerebral hypoxia or by causing a sharp rise in blood pressure.

One explanation of eclampsia is that the metabolic or enzymatic block in the extra cellular fluid compartment of the brain, which normally protects the cortex from afferent stimuli capable of inducing seizures, may be rendered ineffective by the metabolic alterations and compartmental shifts in fluid and electrolytes.

The convulsions in eclampsia are identical to grand-mal epileptic fits. Usually there are only one, or maybe two fits, but occasionally a condition resembling status epilepticus occurs.

The occurrence of eclampsia immediately worsens the prognosis for the baby and the mother, being associated with a 7% maternal mortality.

The differential diagnosis of eclampsia is:

i. Epilepsy,

ii. Toxic response to regional or local anaesthesia.

Causes of Maternal Death in Pre-eclampsia:

a.	Circulatory failure	25%
b.	Pulmonary oedema	25%
c.	Cerebrovascular accident	25%
d.	Renal failure, infection and other causes	25%

Intra-cranial haemorrhage is the most common cause of death in women dying from *eclampsia.*

Assessment, Investigations and Diagnosis of Hypertension on Pregnancy:
History:

During the second half of pregnancy the routine history taken at each antenatal visit should include enquiries into tightness of rings, presence of headaches, drowsiness, blurred vision, or scotoma, Scotomas may occur in normal pregnancy but if they occur with pre-eclampsia they have serious implication, being due to circulatory disturbances in the visual cortex, optic nerve, or retina. Other symptoms of pre-eclampsia include epigastric pain (from distension of the liver capsule), and rapid weight gain. If the patient is putting on excess weight then an enquiry into the diet should be made to exclude overeating as the cause.

Examination:

Accurate weight, blood pressure recordings and urinalysis for protein are routine antenatal proceedures. Blood pressure should be recorded at the beginning of the visit before the patient has relaxed completely as this is a more accurate indication of the patients blood pressure under the stress of normal everyday activities.

Another parameter in pre-eclampsia is weight gain with or without demonstrable oedema. A weight gain of over one Kilogram per week is abnormal especially in the third trimester. It should be noted that swelling of hands and ankles does occur in about 60% of normal pregnancies. Even so, oedema of the fingers and face must be carefully sought for and if it is present, pre-eclampsia must be excluded.

Other findings which arouse suspicion of pre-eclampsia are the presence of hyperreflexia and/or sustained ankle clonus.

At each antenatal examination the doctor must be alert to detect any of the factors pre-disposing to pre-eclampsia.

An accurate estimation of gestation age in the first twelve weeks by bimanual palpation of the uterus is of paramount importance because if pre-term induction is indicated this can be timed with accuracy and the risk of prematurity anticipated.

In a case of diagnosed pre-eclampsia the clinical assessment of uterine size will provide a rough estimate of progress or retardation of foetal growth.

Investigation:

1. If the ward urinalysis indicates that protein is present in a carefully collected mid-stream specimen of urine, this should be evaluated quantitatively by acidifying (with a drop of acetic acid) about 10 mls. of the urine in a test tube and then rolling it. The protien in the urine will be denatured and precipitate out of solution and can be quantitated as percentage of the volume in the urine sample. If there is any doubt as to possible contamination of the sample, a repeat specimen should first be taken after the vulva and vagina have been swabbed with antiseptic solution, then the quantitative measurement of protein may be made.
2. In a case of diagnosed or suspected pre-eclampsia, blood urea and serum creatinine estimations should be made to assess renal function. The upper limits of normal in pregnancy are blood urea 28 mg/100 ml; serum creatinine 2 mg/100 ml.

3. If a patient has pre-ecclampsia then serial urinary oestriol determinations are a useful indication of placental adequency or inadequacy at the 30th week of pregnancy. A fall in values over several days usually indicates placental insufficiency which may lead to foetal death. In such a case, induction of labour should be considered.

4. The other investigations relevent to pre-eclampsia are those aimed at determing foetal maturity when pre-term induction of labour is being considered.

   Echograms estimate the size of the foetal head and trunk when compared to the average foetal size for that gestational age and are of immense value in detecting failure to grow.

   X-rays have limited value in investigating the effects of hypertension on pregnancy for they can usually only detect foetal age when epiphysis are present. The distal femoral epiphysis becomes visible at about the 36th week and the proximal tibial epiphysis at about the 38th week.

Management:

a. *Hypertension not due to Pre-eclampsia*

   The effects of hypertension on the placenta and foetus are similar to the effects of pre-eclampsia so the management follows a similar pattern. However, while anti-hypertensive drugs are avoided in managing pre-eclampsia, the patient with non-pre-eclamptic hypertension should usually stay on her pre-pregnancy drug regime, with the omission of any teratogenic drugs, She should be closely observed for the development of concomitant pre-eclampsia.

b. *Pre-eclampsia*

   Because delivery of the placenta removes the major source of the initiating cause of pre-eclampsia, and because deterioration of placental function may lead to intra-uterine death, early induction of labour is indictaed in the management of pre-eclampsia, the actual timing of induction depending on the severity of the disease. However, in a number of cases, pre-eclampsia develops at such an early stage that delivery of the foetus would severely jeopardise its chances of survivial. Because of the hazards of prematurity an attempt is always made to carry the pregnancy as far as possible

towards term to achieve foetal maturity without in any way jeopardising the foetus whilst it remains in utero.

In cases of pre-eclampsia in which immediate delivery is not indicated *bed rest* with toilet privileges only is the basic treatment. This should be used even if only one of the parameters (e.g. hypertension or oedema) is present to a significant degree. Sedation (Valium 2-5 mg t.d.s. and Mogadon at night) will make bed rest more acceptable and may possibly remove the anxiety component of the hypertensive state. In most cases this form of management will result in a fall of the blood pressure with a diuresis and decrease of oedema.

Hypotensive agents do not result in increased placental perfusion. Moreover, they remove a valuable parameter of the severity of the condition – elevated blood pressure – and so should only be used if a sharp rise in blood pressure leads to the danger of maternal cerebral haemorrhage.

Diuretics are not given as a definitive treatment for pre-eclampsia but may be used to treat the oedema symptomatically.

If the treatment outlined results in a satisfactory control of the symptoms, and if the gestational age is known with accuracy, delivery should be timed for about 37-40 weeks amenorrhoea. If, however, there is evidence (from oestriol estimations) that placental inadequacy is severe, delivery may have to be carried out earlier. In such cases the risk of foetal or peri-natal death from pre-eclampsia must be weighed against the risks of peri-natal death following premature delivery which are as follows:

Risk of Peri-natal death at –

28/52	–	nearly 100% mortality
30/52	–	60% "
32/52	–	30% "
34/52	–	15% "
36/52	–	7% "
38/52	–	3% "
40/52	–	1% "

Treatment of Sharp Rise in Blood Pressure:

If the blood pressure does rise suddenly (e.g. to 190/115) then the immediate risk is that the mother may progress to eclampsia and possibly have a cerebral haemorrhage. In such a case a rapidly acting

antihypertensive (Hyperstat (Diazoxide)) is used. The dose is 300 mg I.V. in 30 seconds. This may be supplemented at ½-3 hourly intervals but the body rapidly becomes insensitive to its action so that it is rarely of use after 24 hours.

Valium up to 100 mg I.V. or Phenobarbital 30-60 mg. I.V. may also be given at this time to reduce the probability of a fit. Such treatment should be followed as soon as possible by *delivery*.

Treatment of Eclampsia:

This is an emergency and initial treatment will probably be given by a nurse. It consists of –

a. Maintaining an airway.

b. Preventing tongue biting (padded spoon handle or Guedel's airway).

c. Administration of oxygen (via face mask).

Definitive treatment consists of –

a. Valium 100 mg. I.V. or Phenobarbital 30-60 mg. I.V. in an attempt to control the fit and prevent further fitting.

b. Delivery by the quickest and least traumatic method as soon as fitting is under control.

Prognosis:

For the mother –

a. *Pre-eclampsia* is the third leading cause of death in obstetric patients (after abortion and haemorrhage). This incidence can be greatly reduced (to almost zero) by proper management but at present is rising in many countries due to increasing complacency and carelessness. It is a reversible disease and does not produce chronic hypertension, chronic renal disease or other sqeuelae.

b. *Eclampsia*

This is very dangerous to the mother but if it is not complicated by a cerebral haemorrhage or death in the acute stage, recovery should be complete after delivery.

Pre-eclampsia is not likely to recur in a patient unless one of the major predisposing factors earlier mentioned is present.

For the baby –

Peri-natal mortality is about 5% in pre-eclampsia if pre-eclampsia is present from the 36th week. It rises to over 50% if eclampsia occurs. An increased incidence of feotal abnormalities and epilepsy has been reported in babies from pregnancies complicated by pre-eclampsia.

The Major Drugs used in the Management of Pre-eclampsia:

1. **Valium** (Diazepam)

Mode of action and dosage : a. "Anti-anxiety" or "Mental relaxation" effect when used in relatively low doses (e.g. 2-5 mg. orally, I.M. or I.V.). Some consider that this may contribute to lowering of the blood pressure by eliminating the "anxiety component" of hypertension.

b. Muscle relaxation is produced by large doses (e.g. 100 mg.) I.V. This is used in the treatment of eclampsia or imminent eclampsia.

Onset of Action : Within seconds of intravenous injection.

Problems : Potentiation by barbiturates and morphine derivatives.

2. **Hyperstat** (Diazoxide)

Mode of Action : Relaxes constricted smooth muscle in the peripheral arterioles thus resulting in a direct decrease in peripheral resistance without interfering with placetal blood blood flow.

Dose : 300 mg. I.V. in 30 seconds.

Onset of Action : Blood pressure usually falls within 5 minutes of administration.

Duration of Action : There is a gradual return of blood pressure to normal value over 4-6 hours. It may be repeated at this time but blood pressure

generally becomes refractory to its effect within 24 hours.

Special Advantages : It does not complicate anaesthesia. No harmful effects on the foetus have been demonstrated.

It is unusual to get hypotension as a result of its use.

It may transiently increase blood glucose levels.

Disadvantages : It may interrupt labour. Rarely it may cause hypotension and shock.
Gastro-intestinal upset

Sodium and water retention (after repeated injections).

3. **Mogadon** (benzodiazepine) 5-10 mgm at night.

Duration of action : 6-8 hours of deep sleep.

Special Advantages : It induces deep sleep and relieves anxiety states without producing depression. It has no apparent harmful effects on the foetus.

CHAPTER 6

BLEEDING IN EARLY PREGNANCY

General Instructional Objective

Understands the significance, causes and treatment of vaginal bleeding in early pregnancy so that he can manage this condition appropriately.

Specific Behaviours

1. Describes the physiology of conception and pregnancy [with relation to implantation.]
2. Describes the various symptoms and signs which may be associated with vaginal bleeding in early pregnancy and their diagnostic significance.
3. Discusses the various causes of vaginal bleeding in early pregnancy.
4. Evaluates the physical condition of women with vaginal bleeding in early pregnancy.
5. Makes and discusses his provisional diagnosis of the cause of vaginal bleeding in women in early pregnancy.
6. Discusses the management of women with vaginal bleeding in early pregnancy.

Bleeding in Early Pregnancy

A. Physiology of Conception and Implanation

Some 9 to 11 days after ovulation, when the blastocyst has become completely buried within the endometrium, the trophoblast is actively

developing two layers – the syncytiotrophoblast, and cytotrophoblast (Fig. 6.1). The syncytiotrophoblast rapidly develops into the primitive placenta, in which primitive lacunae form later to be the intervillous spaces. The lacunae fill with blood under low pressure when a maternal blood vessel is eroded (Fig. 6.1). At this stage the stromal cells of the

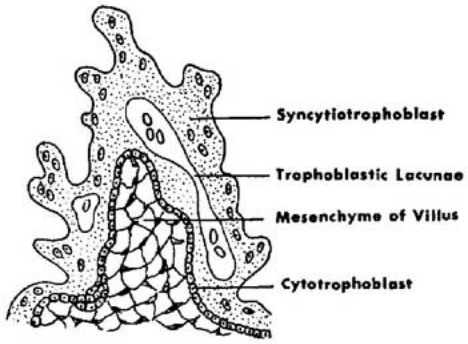

Fig. 6.1. Early stage in the development of chorionic villi. The trophoblast has developed two layers – the syncytiotrophoblast and the cytotrophoblast. The lacunae contain a few maternal red blood cells. The mesenchyme core is developing within the villus. (After Llewellyn – Jones, 1971).

endometrium become polyhedral in shape and fill with glycogen and lipid, supplying the nutritive needs of the trophoblast. This change transforms the *stroma* into *decidua*, and is due to the continuing activity of oestrogen and progesterone.

Further development of the trophoblastic knobs leads to their becoming finger-like projections termed chorionic villi. Most of this growth takes place on the surface nearest the endometrium (Fig. 6.2). setting the stage for the development of a definitive placenta. The lacunae now coalesce and soon the erosion of a maternal artery fills and expands the lacunae with blood under high pressure. Syncytial sprouts now appear on sides of the large villi and by subdivision form a branching structure of free villi within the blood lakes (Fig. 6.3) The blood from the maternal spiral arteries is injected during systole at a pressure of about 80 mm. Hg., bathing the villi. This process allows an exchange of nutrients and for waste products which leave the lacunae in "venous" blood through the decidual plate (Fig. 6.3). The maternal

blood flow through the entire placenta approaches 500-700 ml. per minute near term.

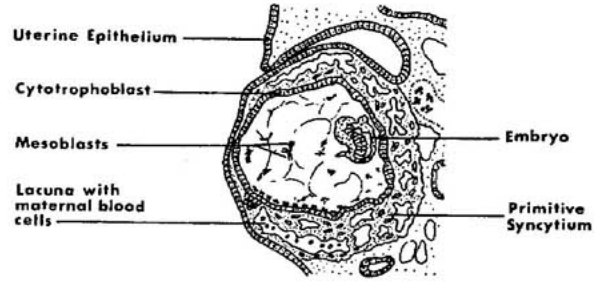

Fig. 6.2. Section of an 11½ day human embryo. The blastocystic trophoblast has differentiated into primitive syncytium and cytotrophoblast. Mesoblast has differentiated from the inner surface of the latter and almost fills the original blastocystic cavity. Lacunae have appeared in the actively growing syncytium, and the maternal blood cells have seeped into several of them. Buds are appearing at intervals on the syncytium. These are the forerunners of chorionic villi. (From Llewellyn–Jones, 1971).

The spurts of blood are intermittent, being dependent on maternal heart as well as uterine contractions (during labour) during which the placental blood flow may be reduced to about 200 mls. per minute. The intervillous space volume may be reduced by fibrinoid deposits and by infarcation. In cases of hypertension and pre-eclampsia reduction in volume may also be contributed to by vascular spasm of maternal vessels.

Placental Exchange

The placenta must assume the function of the kidney, lungs, and intestine on behalf of the foetus. It does so by providing a villous surface of some 14m² for exchange. Transfer of many substances takes place by simple osmotic diffusion due to concentration gradients (e.g. O_2, CO_2 and urea). Some compounds such as amino-acids, are actively transported into the foetal plasma against a concentration

gradient. Pinocytosis can transport larger molecules such as plasma proteins, e.g. IgG.

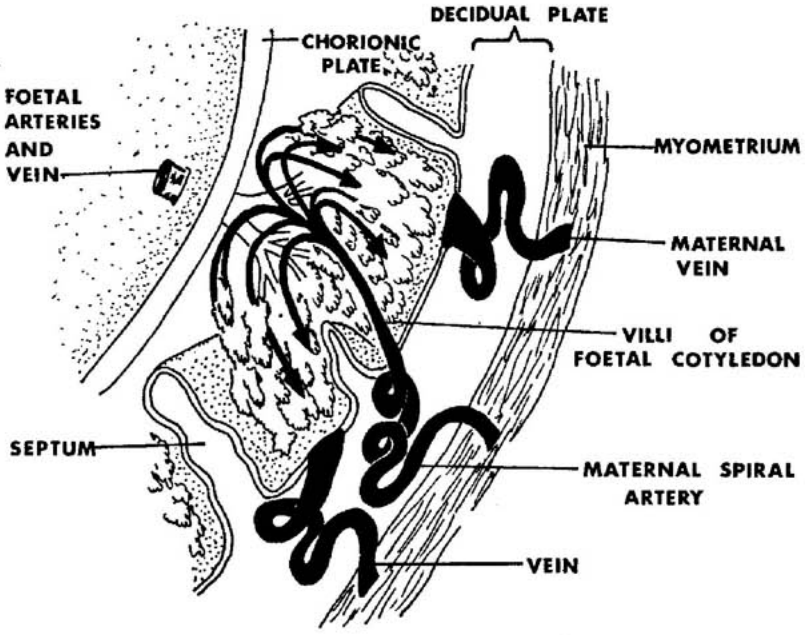

Fig. 6.3. Diagram of an intervillous space. A cotyledon is seen bathed in a fountain of maternal blood. The blood then escapes through maternal veins. (After Llewellyn-Jones, 1971).

B. Symptoms and Signs Associated with Vaginal Bleeding in Early Pregnancy

1. **Pain.** This may be a *colicky*, low abdominal or back pain in threatened, inevitable, and incomplete abortions, and usually follows the bleeding. It is due to uterine contractions reaching such an intensity that cervical dilatation occurs.

   Severe pain may be suggestive of an ectopic pregnancy. (Pain comes before bleeding in some 25% of cases.) Consider also septic abortion.

2. **No Pain.** Early threatened abortion; Hydatidiform mole; Cervical polyp, implantation bleed, ectopic pregnancy.

3. **Passage of Clots.** Associated with heavier bleeding of the inevitable and incomplete abortions and hydatidiform mole.

4. **Passage of 'Grape-like' Structures.** Hydatidiform mole.

5. **Decline of Symptoms of Pregnancy.** With loss of placental function and hormone production in missed abortion.

6. **Tender Abdomen.** *Soft* – threatened, inevitable, incomplete, or septic abortion.

   Guarding – Ectopic gestation, septic abortion with peritoneal soiling due to criminal interference *Tender vaginal fornices* – as above.

7. **Shock** – (pallor, sweating, air hunger, tachycardia, cold skin, hypotension).

   a. *Hypovolaemia* – with inevitable and incomplete abortion, ruptured ectopic gestation, hydatidiform mole.

   b. *Septic* – septic abortion.

   c. *Traumatic* – during dilatation of the cervix in a criminal abortion; or with products of conception lodged in cervical canal.

8. **Uterine Size**

   Consistent with period of amenorrhoea – the threatened, inevitable and incomplete abortion and hydatidiform mole, and bleeding due to causes unassociated with the pregnancy.
   Smaller than period of amenorrhoea – in complete abortion, ectopic pregnancy, hydatidiform mole.
   Larger than period of amenorrhoea – with a hydatidiform mole.

9. **State of the Cervical Os.** *Closed* – always closed in threatened abortion. Significance lies with the open os in, e.g. inevitable and incomplete abortion, and hydatidiform mole.

10. **Pre-Eclampsia and/or Hyperemesis** – associated with the hydatidiform mole.

C. Causes of Vaginal Bleeding in Early Pregnancy

The most important individual conditions will be treated more fully, outlining management of each.

Summary of Causes of Bleeding

1. Threatened Abortion

2. Inevitable Abortion
3. Incomplete Abortion
4. Missed Abortion
5. Complete Abortion
6. Septic Abortion
7. Ectopic Gestation
8. Implantation Bleed
9. Cervical Lesion e.g. infected ectopic columnar epithelium; polyp (see Chapter 18)
10. Vaginal Infection, e.g. trichomonas; gonorrhoea (see Chapter 13)
11. Trophoblastic Tumour (see Chapter 19)
12. Carcinoma (see Chapter 19)
13. Bleeding Diathesis

(A placenta praevia may rarely bleed in the latter weeks of the first half of the pregnancy.)

Abortion

The term "abortion" is synonymous with the term "miscarriage", and the latter is the one that should be used when talking to a patient. (The connotations of the two terms are different to the lay mind, where "abortion" implies deliberate interruption of pregnancy and "miscarriage" implies an occurrence ordained by fate.)

Miscarriage (abortion) is premature interruption of the pregnancy before the twentieth week. After this arbitrary twentieth week a delivery of a dead foetus is termed "stillbirth", which requires a registration according to local laws.

There are five degrees of abortion:

1. Threatened
2. Inevitable
3. Incomplete
4. Complete
5. Missed

All of these may be either septic or non-septic. Aetiologically, abortion may be spontaneous or induced.

Aetiology of Abortion

There are many causes of spontaneous abortion. Certain of these are more often associated with early abortion (first 12 weeks), others with late abortions (12-20 weeks.).

Foetal Abnormalities (chromosomal), and hormonal deficiency, are the major factors in early abortions. Incompetent cervix, local uterine abnormalities (e.g. septa, fibroids), and general maternal disease (e.g. febrile infections, anaemia, syphilis) occur more with later aborions.

1. Threatened Abortion

This is diagnosed by the following pattern of symptoms and signs.

Symptoms:

a. The symptoms of early pregnancy.
b. Vaginal bleeding. This is the first symptom, and may vary in amount from "spotting" of bright blood, to a flow equivalent to menstruation. It is unusual for clots to appear.
c. Pain is intermittent and colicky due to uterine contraction. It is a low abdominal or back pain, and is mild. Bleeding usually precedes the pain.

Signs:

a. The signs of pregnancy.
b. Abdomen is soft and any tenderness being confined to the uterus.
c. Uterine size is consistent with the period of amenorrhoea.
d. The cervix uteri is CLOSED. On inspection a small amount of blood may be seen coming through the os.

Management:

On diagnosing a threatened abortion by the above symptoms and signs, the condition should be explained to the patient. In about 70% of threatened abortions the pregnancy will continue. The bleeding will not damage the foetus. but if it is abnormal, the pregnancy will miscarry. Explain that the cause does not lie with any actions which the patients have taken.

Treatment includes:

a. *Bed rest* at home. Patient is confined to bed until 1-2 days after cessation of bleeding. Intercourse should be avoided until there has been no bleeding for one week.
b. *Sedation* – An initial dose of a narcotic such as Pethidine 100 mgm should be given. Subsequently, daily sedation may be achieved with Valium 5 mgms. t.d.s. plus Sodium Amytal

100-200 mgms at night if necessary. There is no proof that any type of hormone is of any help in these cases.

2. **Inevitable Abortion**

Symptoms:

a. Symptoms of pregnancy.
b. Vaginal bleeding. Here bleeding tends to be heavier than in cases of threatened abortion, and may be excessive with the passage of clots.
c. Pain is a more prominent symptom than in threatened abortion. Usually it follows the bleeding and its distribution is similar to that of threatened abortion and is due to uterine contractions reaching an intensity sufficient to produce cervical dilatation.

Signs:

a. The signs of pregnancy.
b. The signs of shock and collapse if the bleeding has been excessive.
c. Abdominal signs are similar to those of threatened abortion.
d. Uterus is of size consistent with the period of amenorrhoea.
e. The cervix uteri is *dilated* and will usually admit a finger. The products of conception can be felt through but *not* in the cervical canal.

Management:

On diagnosing the condition the situation is explained to the patient. Although called 'inevitable' it may rarely settle down and continue so that treatment is basically conservative (as for threatened abortion), until the inevitable abortion converts to an incomplete abortion when definitive steps are taken. (This process is usually only a few hours in duration).

The patient is admitted to hospital where the extent of the bleeding can be monitored. Blood is taken early for cross matching. Conservative treatment is abandoned if the patient continues to bleed to any degree. The uterus is evacuated in theatre before the patient becomes shocked from cervical dilatation or is exsanguinated. If this had not been done previously in the antenatal clinic, the patient's blood group is determined and if *Rh* negative, 200 mg (1ml) of anti-D gamma globulin are given intramuscularly within 48 hours of the curettage to reduce the chance of maternal isoimmunization. (see Chapter 8).

3. **Incomplete Abortion**

Symptoms:

a. Symptoms of pregnancy.
b. Vaginal bleeding. Here again bleeding can be heavy and on occasions alarming. It is accompanied by the passage of clots and often recognizable products of conception, which may be the gestation sac, the foetus, or part of the placenta.
c. Pain may be lower abdominal or back pain, which is colicky, and increases with the passage of clots or products of conception. It follows the onset of bleeding.

Signs:

a. The signs of pregnancy.
b. Signs of shock and collapse, depending on the degree of blood loss, may be present, distention of the cervical canal by placental debris may also cause hypotension by reflex nervous stimulation.
c. Abdominal signs are those of inevitable abortion.
d. Uterine size is usually the same as that anticipated for the period of amenorrhoea.
e. The cervix is dilated and the canal will admit a finger. Products of conception may be present in the canal, protruding through the cervical os, or lying in the vagina. This is a picture of a recent incomplete abortion. If the abortion has occurred a few days, or even weeks before the patient is seen, the cervix will no longer admit a finger, although it is usually patulous, and the uterus may be enlarged to very little beyond normal size. In these cases the blood loss tends to be dark and may be offensive.

Management:

After diagnosis and explanation, the patient should be *admitted* to hospital and *resuscitative* measures instituted if necessary. If in severe shock an intravenous infusion of albumin and saline, plasma, or S.P.P.S. solution should be commenced while waiting for the carefully cross matched blood. Blood group is determined as in the case described under "inevitable abortion".

A *vaginal examination* must be performed to establish the diagnosis. If products of conception are present in the cervical canal they should

be removed gently, either digitally or with ovum forcepts. This often improves the patient's condition and may diminish the bleeding. Now an *injection of 0.5 mg ergometrine* is given intramuscularly, or intravenously if the patient is shocked.

With the patient fully resuscitated *surgical evacuation* of the uterine cavity is carried out. If emergency measures have controlled bleeding there are some advantages in delaying evacuation for 6-12 hours. These include,

a. a fully resuscitated patient with an empty stomach,
b. contracted and retracted uterus less likely be to perforated during evacuation.

Evacuation of the uterus is carried out aseptically in an operating theatre under general anaesthesia. The bladder is emptied by catheterization, a bimanual examination confirm the position and size of the uterus, and excludes other pathology. If necessary, the cervix is dilated and the uterus evacuated by ovum forceps and a large blunt curette. Before evacuation is commenced 0.5 mg ergometrine is given intravenously.

4. Complete Abortion

The history is similar to that of an incomplete abortion, but in addition the pain and bleeding should rapidly subside after the abortion occurred. If the products of conception are seen they appear to be complete. This is a rare condition.

On examination the uterus is smaller than would be expected for the period of amenorrhoea, and the cervical canal is empty.

If the woman is seen soon after abortion she should be managed as for incomplete abortion, unless beyond 18-20 weeks gestation, when one can be reasonably certain that a placenta appears complete. Fifty percent of alleged complete abortions occurring before week 18 of gestation will have retained products of conception which may become infected or give rise to heavy bleeding at times. In other words, an early abortion is diagnosed to be complete only when evacuation of the uterus shows that it is empty. When a patient presents with a history of 'complete' abortion days or weeks before, and vaginal loss has ceased or become minimal, examination may show a small uterus and a closed cervix. Under these circumstances no intervention is required.

5. Missed Abortion

A missed abortion is an uncommon condition in which the foetus has died, the pregnancy ceases to progress, and the products have been retained in the uterus. This may follow a threatened miscarriage, or an intra-uterine bleed, which was not accompanied by vaginal bleeding. In such cases the uterus contains blood clot which is fleshy and partly organised, and is sometimes referred to as a carneous mole.

Diagnosis—may be distinguished from a viable pregnancy. The history is one of a variable period of amenorrhoea which may have been punctuated by an episode of bleeding. The symptoms of pregnancy have since subsided. Intermittent slight bleeding or a dark brown discharge are common.

On examination the uterus is *smaller* than expected by dates, feels *firmer* than a pregnant uterus, and the cervix uteri is closed. A pregnancy test is negative. A few weeks later the uterus is still the same size.

Management:

Most women with a missed abortion expel the foetus spontaneously within 6 weeks of the intra-uterine death. By the time diagnosis has been made some weeks have passed, and so non-intervention leading to spontaneous abortion is the best management. Dead tissue is mechanically difficult to remove, and risks of haemorrhage and infection are high.

While awaiting the spontaneous expulsion an intermittent brown discharge may appear, the woman's emotional reaction to a retained dead 'baby' may arise, and the rare problem of hypofibrinogenaemia may occur (see Chapter 7). If, therefore, after 4 weeks of waiting the uterus has not emptied, or as the above complications dictate, active intervention may be decided upon.

With the uterus less than 12 weeks in size it may be emptied by dilatation and curettage. This may be technically difficult as placental tissue may be adherent to the uterus. When the uterus is larger than 12 weeks a syntocinon drip should be commenced and increased rapidly until contractions occur, or until a concentration of 100 u/l is reached. Following expulsion evacuation is completed surgically. In some centres intra-uterine prostaglandins are available as an alternative to syntocinon-induced evacuation.

6. Septic Abortion

Any degree of abortion may become septic (infected). The aetiology is usually either *criminal interference* to procure abortion, or an *incomplete abortion* which has been protracted and infected.

Symptoms and Signs:

a. Pyrexia.
b. Offensive vaginal discharge.
c. A tender uterus.
d. Tachycardia.
e. Signs of extrauterine spread. In 40% of cases infection is limited to the products of conception. In more advanced cases infection may involve the endometrium and myomtrium. It may spread to the pelvic organs as pelvic cellulitis, salpingo-oophoritis, or pelvic abscess. Infection may spread further causing general peritonitis, or even septicaemia and pyaemia.

Special Types:

a. *Clostridium welchii Septicaemia* – The patient is extremely ill and often hypotensive from adrenal failure. If she survives beyond this stage jaundice will set in from haemolysis, and the urine will be port wine coloured as a result. Anuria from renal failure is a late complication.
b. *Endotoxic Shock* – Gram negative bacilli (e.g. E. coli) may bring about profound shock and death due to endotoxin production.

Management:

After diagnosis is reached:

a. Admit patient to hospital.
b. Take cervical cultures (aerobic and anaeorobic). If necessary, blood cultures may be taken.
c. Commence antibiotics. Commence on pencillin 1,000,000 units 6th hourly, and streptomycin 0.5 gram b.d. intramuscularly. Adjust antibiotics according to sensitivity results *and* clinical response. Ampicillin and kanamycin may be necessary in a seriously ill, hypotensive patient.
d. Correct the anaemia by transfusion if haemoglobin is below 9 grams %.
e. Treat hypovolaemic shock by transfusion. With adrenal failure due to Clostridium welchii septicaemia or endotoxic shock large doses of I.V. hydrocortisone (40 mg/kg per 24 hrs. 60-70% of total initially, remainder at 6th hourly intervals) are required.
f. With clostridial infections energetic treatment is required. If infection with these is apparent clinically 1,000,000 units of penicillin 4th hourly are given with combined antigas gangrene serum (75,000 units) 4th hourly.
g. *Evacuation of the uterus* is carried out in 24-36 hours if infection is confined to the uterus on clinical grounds. If extrauterine spread of infection has occurred, conservative treatment with antibiotics should continue, unless surgical intervention is required to control bleeding.

Sequelae – Septic abortion may result in persisting infection as a chronic salpingo-oophoritis causing ill health. The more usual result is tubal damage or occlusion with subsequent infertility.

Habitual Abortion

This is included here for the sake of completeness. The term 'habitual abortion' is applied to a woman who has had *3 or more consecutive spontaneous abortions*. The aetiology is the same as for all abortions. However, a thorough investigation should be carried out to exclude remediable conditions such as a septate uterus or incompetent cervix. These causes are more often absent, than present. When *no* cause is found treatment can be only empirical, including reassurance, avoidance of intercourse in the early months, plenty of rest, and avoidance of heavy work.

7. Ectopic Pregnancy

Definition:

Ectopic pregnancy includes all cases of pregnancy where the fertilised ovum is *NOT* implanted in the endometrium lining the normal uterine cavity.

Incidence:

Varies between 1/100-1/1000 deliveries, being related to frequency of pelvic infections in the community.

Varieties:

A. Extra-Uterine

1. Tubal Pregnancy – this is far more common than all others combined. It may be ampullary, isthmic, interstitial and fimbrial in decreasing order of frequency.
   Aetiology:
   a. Abnormalities of the tube delaying passage of ovum.
      i. Previous inflammatory disease causing:
         . peritubal adhesions *kinking* or *compressing* the tube
         . diminished peristalsis
         . loss of cilia
         . intratubal adhesions
      ii. Developmental abnormalities of the tube:
         . tortuosity
         . diverticula
         . accessory ostia
      iii. Kinking and distortion due to external pressure due to tumours, cysts and fibromyomata
      iv. Spasm of the tube
   b. Increased receptiveness of tubal muscosa to the fertilized ovum, e.g. with endometriosis of the tube. This is a rare cause.
   c. Over-development of the ovum with early implantation.
   d. No obvious cause.
2. Ovarian Pregnancy – extremely rare.
3. Abdominal pregnancy – also rare. Usually this is secondary to a primary tubal implantation. The foetus almost always dies.
4. Intraligamentary pregnancy – secondary to tubal pregnancy.

B. Uterine

1. Cervical pregnancy – rare. Abortion following profuse bleeding may occur.
2. Cornual pregnancy – ovum is implanted in a rudimentary horn of a bicornuate uterus.

Pathology:

In the *uterus* generalized enlargement due to vascularity and muscle hypertrophy seldom exceeds that of an 8 week gestation. The decidual

reaction in the endometrium occurs normally, and is shed in fragments or as a whole when the pregnancy is overcome by some accident. The *fallopian tube* shows some decidual reaction. The trophoblast invades and even penetrates the muscle and blood vessels causing bleeding into and outside the tube.

Sequelae of Tubal Pregnancy

1. Spontaneous regression and complete absorption.
2. Internal tubal rupture may lead to:
   a. Tubal carneous mole formation; due to repeated chorio-decidual haemorrhages causing death of ovum.
   b. Tubal abortion – the ovum is extruded through the abdominal ostium into the peritoneal cavity leading to:
      . intraperitoneal haemorrhage
      . peritubal haematocoele
      . pelvic haematocoele
3. External tubal rupture. The tubal wall may so distend that it will rupture. This may be sudden or gradual and may lead to:
   a. intraperitoneal rupture with:
      . intraperitoneal haemorrhage
      . paratubal haemoatocoele
      . pelvic haematocoele
      . secondary abdominal pregnancy
   b. extraperitoneal rupture with:
      . intraligamentary haematoma
      . intraligamentary pregnancy

   The foetus is often malformed and death is almost inevitable, usually before 6 weeks gestation. Rarely a secondary abdominal pregnancy may survive to term. The dead foetus may become:
      i. completely absorbed
      ii. mummified (adipocere formation)
      iii. calcified, forming a lithopaedion
      iv. infected

Clinical Manifestation:

Tubal pregnancy may present as an acute, or a more chronic illness. The chronic presentation is more common:

a. *The Chronic Presentation*

   This is seen with small but recurrent intraperitoneal bleeding from the tube.

There is a short period of *amenorrhoea* of 3 to 11 weeks (shorter, 4-6 weeks with isthmic, and longer, 12-16 weeks, with interstitial and cornual pregnancies). The *symptoms of pregnancy* are present. *Lower abdominal pain* is the most common symptom and may be due to tubal colic or distension, peritoneal irritation by blood, or uterine contractions expelling pieces of the decidua. *Vaginal bleeding*, almost always due to decidual shedding, is also a common symptom, but its relation to the onset of pain is not helpful diagnositically Bleeding tends to be continuous. Faintness, nausea, epigastric and shoulder pain may also be present.

On examination the patient may be *unaffected or in varying degree of shock* with pallor, tachycardia, hypotension, sweating and cold extremities. The temperature may be slightly raised, normal, or depressed with a massive bleed. *Signs of pregnancy* may be present in the breasts. *Tenderness and guarding* over the lower abodmen is the striking feature. If haemoperitoneum has been present for 2-3 days a blue discoloration of the umbilicus (Cullen's sign) may appear. A tender *mass* in the lower abdomen with indefinite border and with reasonance over its upper portion is characteristic of a pelvic haematocoele.

Tenderness in the vaginal fornices, especially on the affected side, is the most constant sign. Moving the cervix may elicit pain. (This is known as the exquisitely tender cervix of pelvic peritonism). A *soft, tender and irregular mass* may be felt in one or more fornices. The *uterus is only slightly enlarged.* The only helpful laboratory investigations are a falling haemoglobin, a rising bilirubin content, and a rising leukocyte count. A single haematological reading is therefore of little value. Pregnancy tests may or may not be positive.

b. *The Acute Presentation:*

This occurs with a sudden, massive intraperitoneal haemorrhage, typical of tubal rupture. It may complicate the chronic presentation or be the first sign of illness.
Again after a short period of *amenorrhoea* the patient is seized with a severe lower abdominal pain, followed immediately by profound collapse with loss of consciouness in many cases.

On examination the signs of *shock* are obvious (pallor, sweating, hypotension, cold skin, weak rapid pulse, tachycardia, air hunger, and subnormal temperature). The *abdomen is again tender* and there may be dullness to percussion in the flanks. If vaginal examination can be carried out, the signs described above will be present.

Differential Diagnosis:

a. Abnormalities of a uterine pregnancy:–
   - Abortion – all degrees
   - Early intrauterine pregnancy complicated by intestinal or renal colic or by a pelvic tumour, e.g. ovarian cyst or fibromyoma

b. Conditions causing abdominal pain:–
   - Salpingitis or salpingo-oophoritis
   - Dysmenorrhoea
   - Torsion of ovarian cyst or subserous fibromyoma
   - Appendicitis
   - Perforated peptic ulcer
   - Renal colic
   - Pyelonephritis

c. Conditions causing intraperitoneal haemorrhage:–
   - Rupture of a Graafian follicle or corpus luteum
   - Rupture of spleen or liver
   - Ruptured endometriotic cyst

d. Conditions simulating a pelvic haematocele:–
   - Retroverted gravid uterus
   - Tubo-ovarian or pelvic abscess

Management:

The diagnosis of ectopic pregnancy is often difficult to make. On history and examination therefore, *suspect* it and search for all relevant symptoms and signs if the situation allows. Admit to hospital. Take blood early for cross matching, blood count, and *Rh* grouping. Set up an I.V. drip with saline or S.P.P.S., depending on the condition of patient. The patient will require morphine or pethidine early, but be sure of the diagnosis before blunting the signs and symptoms with drugs. Arrange for a laparotomy.

In the chronic presentation, *when diagnosis is difficult to make*, observe the patient keeping an hourly chart on B.P., pulse and temperature, until a firm diagnosis is made. This procedure will eliminate four out of five unnecessary laparotomies.

In the acute presentation treat shock primarily, with rapid fluid replacement followed by blood. Arrange for immediate laparotomy.

At operation a *unilateral salpingectomy* is by far the most common procedure when the other tube and ovary are normal. *Salpingotomy* is performed when the other tube is not normal. *Hysterectomy* is indicated in interstitial pregnancy if there are sufficient children, or if the uterus is diseased.

8. **Implantation Bleed**

This corresponds to the release of some maternal blood during implantation of the ovum. Bleeding occurs earlier than the expected period, and is scanty.

Other causes of bleeding in early pregnancy listed on page 115-116 will be discussed in the appropriate chapters.

Steps in diagnosing the cause of vaginal haemorrhage in early pregnancy:

In diagnosing the patient with vaginal bleeding in early pregnancy one needs to be sure that one is not dealing with rectal or urethral bleeding. Next, the site and cause of bleeding, whether uterine, cervical, or vaginal, will be determined by history and examination. In the history information apart from symptoms may be helpful. The period of amenorrhoea, past pelvic infections or surgery with respect to ectopic gestation, intercourse prior to the onset of bleeding, and any history of attempted instrumental interference, may suggest the diagnosis.

Both general and pelvic examinations are performed. The patient's general appearance, blood pressure, temperature, pulse rate, presence of finger oedema or proteinuria, abdominal tenderness, and fundal height are noted before progressing to the vaginal examination. Using a bivalve speculum the *site* of bleeding is searched for. Most commonly, blood will be seen oozing from the cervical os, although injured ectopic columnar epithelium, cervical polyps or a carcinoma may sometimes be seen bleeding lateral to the os.

A *gentle* bimanual examination will help determine the size, consistency, and position of the uterus, the consistency of the cervix, and any associated masses.

Once the site of bleeding is known and a *differential diagnosis* determined some investigations may need to be ordered. Cervical swabs (infection), cervical biospy (carcinoma), echogram (tropho-blastic tumour; placenta praevia), blood levels of H.C.G. (trophoblastic tumour), and coagulation studies (bleeding diathesis) are some examples.

The extent of the abovementioned procedures will vary with the patient's specific condition. Some causes of haemorrhage will be diagnosed on history and examination alone, while others will require investigations.

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CHAPTER 7

BLEEDING IN LATE PREGNANCY

General Instructional Objective

Understands the cause and treatment of bleeding in late pregancy so that management can be indicated.

Specified Behaviours

1. Assesses women with bleeding in late pregnancy to determine the physical condition and cause of the bleeding.
2. Discusses critically the management of women with bleeding in late pregnancy.
3. Participates in the management of post-partum haemorrhage.
4. Explains the aetiology of bleeding in late pregnancy and discusses prophylaxis.
5. Explains pharmacology of drugs that may affect post-partum haemorrhage.
6. Competently counsels women with antepartum haemorrhage.

Bleeding in Late Pregnancy

In this chapter consideration will be given to bleeding from the genital tract of a pregnant woman after the 20th week of pregnancy and prior to the delivery of the infant. This condition is commonly referred to as *antepartum haemorrhage*.

The common causes of antepartum haemorrhage are:

1. Placenta praevia.
2. Accidental haemorrhage (abruptio placentae).
3. Lesions of the cervix, vagina or vulva, such as polyps, carcinoma, infections, varices or trauma.
4. Vasa praevia.
5. A "show" at the beginning of labour.
6. Bleeding of unknown origin.

1. Placenta Praevia

Placenta praevia is that condition which occurs when the placenta is partially or wholly implanted in the lower uterine segment.

Anatomy:

Four types are described based on the anatomical position, and these are illustrated in Fig. 7.1. The placenta is usually larger in area, but thinner when implanted low in the uterus. Another abnormal finding related to the relative absence of decidua is a deeper penetration of trophoblst into myometrium giving a form of placenta accreta. Velamentous cord insertion is more common and is associated with vasa praevia.

Incidence:

Placenta praevia occurs in from 1-1.5% of all pregnancies which are delivered after 28 weeks.

Aetiology:

The causes are *unknown* but it is thought in part to be related either to fusion of the decidua capsularis with the decidua vera or to extension of the villi at an early stage due to defective decidual reaction.

Clinical Symptoms and Signs:

The typical symptom is of a painless, apparently causeless vaginal haemorrhage occurring during the last trimester of pregnancy.

Almost certainly there will be repeated episodes of minor blood loss which the patient regards as insignificant and which may in fact be ignored by the obstetrician. An antepartum haemorrhage should be viewed with suspicion. About 10% of cases of placenta praevia will be found accidentally when some other obstetric manoeuvre is being performed.

Bleeding may be of small or massive volume and the patient's general condition will reflect the amount of blood lost.

As labour begins the shearing effect of the myometrium and decidua separating from the placenta causes massive haemorrhage. The closer a patient is to going into labour the greater the volume of blood loss. When in labour, the bleeding usually continues.

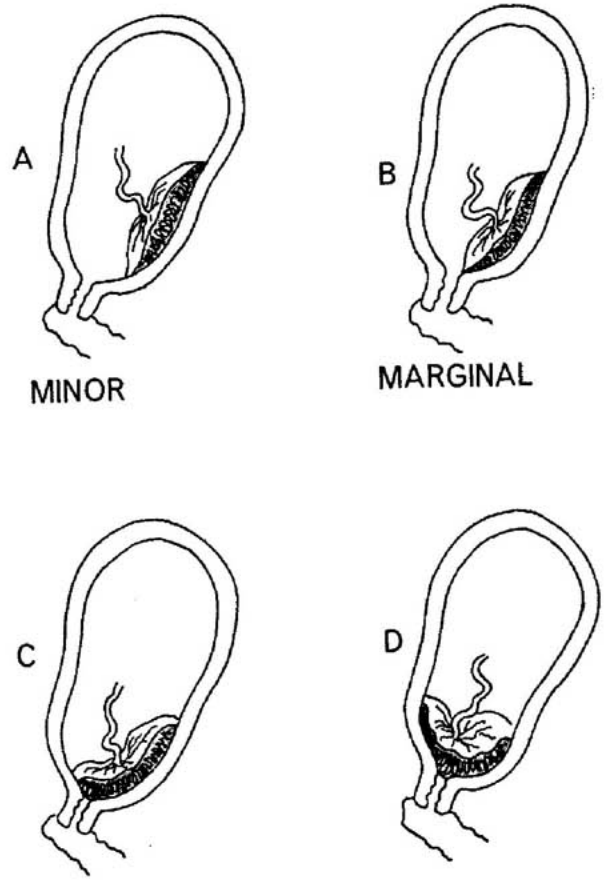

Fig. 7.1. Anatomical classification of placenta praevia.
A. Type 1 – A minor portion of the placenta lies below the lower uterine segment.
B. Type 2 – Most of the placenta lies below the lower uterine segment but the placenta does not cover the internal os.
C. Type 3 – The placenta covers the internal os when the os is closed but not when dilated.
D. Type 4 – The placenta completely covers the internal os even when dilated.

Unstable lie of the foetus and abnormal presentation occur in about 30% of placenta praevias. Obviously if the placenta is well down in the lower segment. the presenting part is prevented from engaging and a breech, transverse lie or high non-engaged head results.

Diagnosis:

Definite diagnosis can only be made by seeing that the placenta is in the lower segment at operation, or by feeling placental tissue vaginally. A clinical diagnosis, however, is made on history and examination. A painless, apparently causeless, repeated bleed should make one suspiscious of placenta praevia. The absence of signs of pre-eclamptic toxaemia (which may be seen if accidental haemorrahge has occurred) may be helpful. The uterus is usually relaxed and soft, and no areas of tenderness can be found.

Using a *Sims* speculum a *careful examination* to determine the site of blood loss is performed. The bleeding may be seen to issue from the os. If from a lesion on the cervix, this can be treated as necessary.

Do NOT perform a digital vaginal examination.

An unstable lie or non-engaged presenting part may make the obstetrician suspect a placenta praevia, especially if associated with a bleed.

Aids to Diagnosis.

1. In expert hands soft tissue *placentography* may be of help. Displacement of the head and an inequality of the thickness of the uterine wall in one place are suggestive. Placentography is of value only after the 35th week, and then is only 80% accurate.
2. *Isotope placental localision* (available at the Royal Hospital for Women) is about 97% accurate. and is based on increased radioactivity counts over the placenta after injection of material such as I^{132} or Technesium 99.
3. *Ultrasonic Placentography* is about as accurate as the isotope method, but is quicker and free of complications.

Management:

The patient with a suspected placenta praevia should be *transferred to a fully -equipped hospital* where *bed rest* is advised until a diagnosis is made. Two units of blood are *cross matched* and stored for any urgent transfusion. Occasionally, transfusion may need to be carried out immediately if the bleeding has been severe. Once in hospital, the further management is either *active* or *expectant.*

a. *Active Management*

This is generally only carried out after the 36th week, or when the patient continues to bleed profusely. Continuing haemorrhage necessitating interference is usually associated with the onset of labour.

If, after the 36th week, a decision has been made for active interference, what should be done?

In an operating theatre fully equipped for Caesarean section, and the patient fully anaesthetised, a careful vaginal examination is performed. First, the fornices are carefully palpated to determine if possible, whether a placenta is lying in the lower segment. If no placenta is felt, a finger is passed gently through the cervical os to feel the membranes, placenta or foetal presenting part. If no placenta is felt, the membranes are artificially ruptured and the patient allowed to go into labour.

If a type III or IV (Fig. 7.1). placenta praevia is discovered, the management is to perform a Caesarean section. A type II placenta praevia of posterior position may necessitate a Caesarean section, but if lying in front should not obstruct delivery. A type I placenta praevia should allow vaginal delivery unless bleeding continues.

b. *Expectant Management*

As large majority of patients with suspected placenta praevia are first seen before they reach 36 weeks gestation, active treatment would necessitate delivery of a large number of premature infants if all patients were treated in this manner. For this reason, expectant management was introduced by Macafee of Belfast in the early 1940's. He treated all his cases of placenta praevia who presented before the 36th week by bed rest. As a general rule these patients rarely suffered a lethal haemorrhage at the first bleed. By putting them to bed and keeping activity to a minimum, the chances of a further placental separation are very much reduced.

Blood should be cross matched and kept stored at all times.

An attempt should be made to increase the haemoglobin level by iron thereapy (see Chapter 211).

At 38 weeks of amenorrhoea, an examination under anaesthesia is performed to assess the stage of placenta praevia, and either the

membranes ruptured or a Caesarean section performed. It is imperative that these procedures be performed in an operating theatre with full Caesarean section set-up.

2. **Accidental Haemorrhage** (Abruptio placentae)

Definition:

Accidental haemorrhage is retroplacental antepartum bleeding which occurs from a normally situated placental site after the 20th week of gestation. External bleeding may occur from this site if the placenta is detached at the edge.

Incidence:

Accidental haemorrhage occurs in 0.7 -1% of pregnancies.

Pathogenesis:

The placenta separates through the decidua allowing blood to "lake" under the placenta. As the blood collects under the placenta it may strip up more placental tissue or force its way between the membranous chorion and the decidua till it reaches the cervix, and then appears as revealed blood loss (Fig. 7.2 - A,C). If the retroplacental haemorrhage strips up the placenta and does not escape externally, a concealed accidental haemorrhage results (Fig. 7.2 B). A small concealed bleed may organise, become fibrosed and calcified producing a corresponding area of infarction and fibrosis in the placenta itself.

With a recent bleed an area of depression with an attached clot may be seen on the surface of the placenta at delivery. If some time has elapsed, infarction at varying stages of organisation will be present.

Grades of Accidental Haemorrhage:

Accidental haemorrhage may be:

a. Mild – 80% cases – less than $\frac{1}{4}$ separation of placenta
b. Moderate – 15% cases – $\frac{1}{4}$-$\frac{2}{3}$ separation of placenta
c. Severe – 5% cases – more than $\frac{2}{3}$ separation of placenta

This classification is based on the amount of placental involvement and the resulting shock which follows.

If the placental area involved is small and under a quarter of the placental site affected, the condition is generally of mild degree, and foetal prognosis is generally good. The blood strips the chorion from the decidua and trickles out through the os to appear as a revealed accidental haemorrhage. As the area of placenta involved is small, blood loss can be replaced. A foetus can exist with about one third of the normal placenta affected, obtaining sufficient nutriment and oxygen through the remaining placenta.

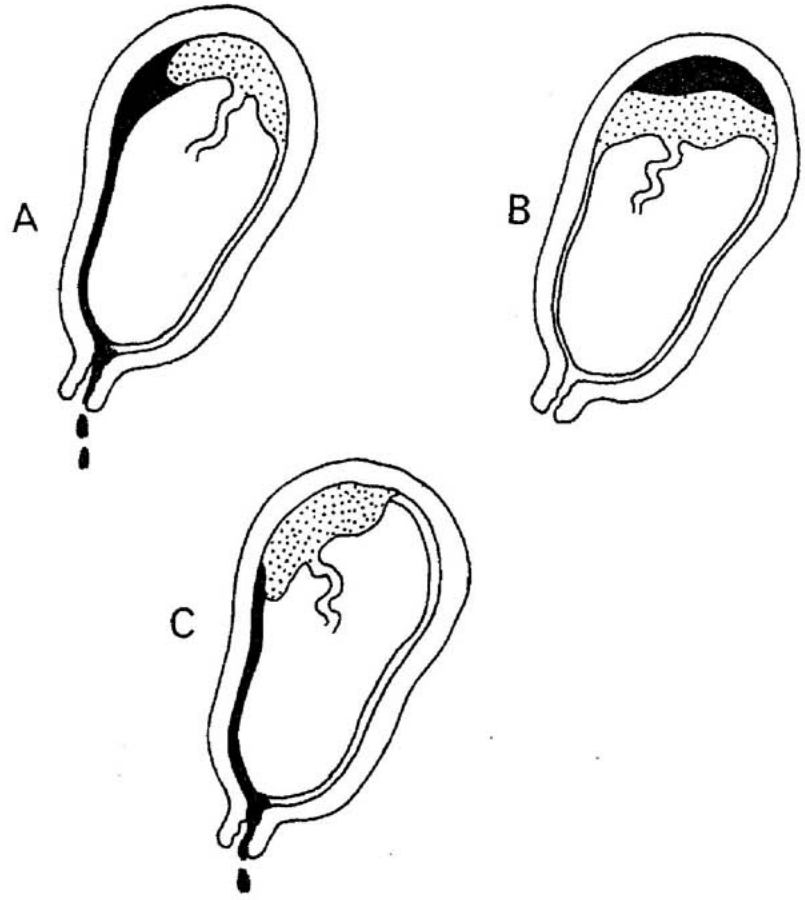

Fig. 7.2. Degrees of detachment of a normally implanted placents.
A. Retroplacental haemorrhage with partial detachment and external bleeding.
B. Concealed haemorrhage. The placental margin has remained attached to the uterus allowing no external bleeding.
C. A small retroplacental haemorrhage with marginal detachment and external bleeding. (Redrawn from Danforth, 1971).

If over a quarter of the placenta is separated, the blood loss tends to be greater, more placental tissue is rendered hypoxic and the foetus

is at greater risk. There is generally some delay in seeing vaginal blood loss after the initial onset of symptoms, and this may be recorded as concealed/revealed accidental haemorrhage.

In those cases where the blood first strips the whole of the placenta from the decidua, there is a considerable collection of blood behind the fully separated placenta. Shock may be profound, foetal death always occurs and bleeding from the vagina may not be seen.

In the moderate to severe forms of accidental haemorrhage, there may be a gradual filling of the uterus with blood. The blood spreads through the decidua and myometrial tissue and separates muscle bundles. This blood may track into the broad ligaments and to the serosal surface of the uterine wall. Because of the blood in the myometrium of the uterus and muscular irritability, this organ usually becomes tense and tender and later goes into spontaneous labour.

Aetiology and Associated Factors:

The actual aetiology of accidental haemorrhage is not entirely settled but there are a number of conditions which are found to be associated with its occurrence:–

a. *Multiparity* – There is a rising incidence of accidental haemorrhage with increasing parity, and a woman having 5 or more children has a four-fold increase in the risk of suffering from accidental haemorrhage over the primipara.

b. *Age* – There is a slightly greater risk with increasing age but this appears to be closely related to increasing parity in the majority of cases.

c. *Nutrition* – People from lower income brackets who eat less protein and green vegetables have a higher incidence of accidental haemorrhage than women in the upper social classes.

d. *Anaemia*

   i. Some 20% of women who had accidental haemorrhage were found to have a haemoglobin below 11.0 gm % before the onset of the haemorrhage. A majority of these women were suffering from iron deficient anaemia.
   ii. An interesting fact wihch has recently been observed by Hibbard in England is that 98% of patients who had accidental haemorrhage had a raised F.I.G.L.U level in the urine. Formimoglutamic acid is produced when folic acid is absent from the metabolic breakdown process of histidine to glutamic acid. When bone marrow aspirations were examined magaloblastic erythropoiesis was noted in about 60% of cases suffering from accidental haemorrhage. These observations suggest that low folic acid levels are related to the onset of the accidental haemorrhage.
   This may be due to the fact that folic acid is necessary for normal development and implantation of the placenta into the decidua and that a deficiency leads to faulty attachment of the placenta.
   Folic acid is essential as a co-enzyme in the synthesis of nucleic acids and the requirements of folic acid are therefore related to the extent of cell division and new tissue formation. It becomes obvious then that as pregnancy advances folic acid is utilised at a rapid rate and that stores and absorption cannot keep pace with metabolism.

e. *Obstetric History*

   i. Previous perinatal mortality in patients who suffer from accidental haemorrhage is about four times as high as expected. In other words, these mothers have a bad previous obstetric history, and about one fifth of mothers lose a pregnancy due to appararently unrelated causes.
   ii. Prematurity in other pregnancies is also twice as high in mothers who have accidental haemorrhage as in the overall population.
   iii. The risk of repeated accidental haemorrhage is five times as great in patients who have had an accidental haemorrhage as it is in the overall population.
   iv. Premature Labour – over half the cases of abruptio placentae occur before the 36th week, but the peak number occurs during the 36th week.
   About 40% of all cases of abruptio are in labour when first seen and there may be a precipitating cause for the abruptio in uterine contractions. It might be that uterine contractions, either of premature labour or Braxton Hicks type, acting on an insecurely anchored placenta, may initiate a separation of a minor or major degree.
   Some 90% of the infants born to mothers having acci-

dental haemorrhage are smaller than the size expected for the period of gestation.

f. *Pre-Eclampsia and Hypertension*

Although about 30% of patients who have accidental haemorrhage are found to have a raised blood pressure and proteinuria after haemorrhage, this is thought to be due to compensatory mechanism to the blood loss, leading to vasoconstriction and renal ischaemia. In large groups of pre-eclamptics carefully observed in antenatal and hospital clinics, there is a slight increase only in the number of accidental haemorrhages occurring.

g. *Trauma*

Trauma may very rarely be a cause for accidental haemorrhage, but generally there is no antecedent history of blows or knocks to the abdomen.

h. *Hydramnios*

There appears to be a slightly higher risk of accidental haemorrhage occurring with hydramnios than in the normal population but the majority of accidental haemorrhage cases have no evidence of excess liquor.

i. *Congenital Malformations* are 3 times as common in those cases having abruptio as in normal pregnancies.

j. *Severe Foetal Respiratory and Cardiovascular Disease* was seen five times more often in cases of abruptio placentae as in the overall population.

k. *Renal infection* may be related to deciduo-placental thromboses and precipitation of ischaemic changes leading to accidental haemorrhage.

The clinical impression concerning the aetiology and pathology of accidental haemorrhage is one of inferior social status, poor nutrition and prevalence of anaemia associated with muliparity, recurrent accidental haemorrhage and previous poor obstetric history. The positive fact that folic acid deficiency is particularly common suggests that this may play an extremely important role in the aetiology of abruptio placentae.

Symptoms and Signs:

The severity of the symptoms and signs is related to the amount of placental separation, so that clinically a fair indication may be gained of the grade of haemorrhage.

Mild Antepartum Haemorrhage:

a. *Pain* – a vague lower abdominal discomfort may or may not be present. Here differentiation from a placenta praevia is required.
b. *Tenderness* – Tenderness may be very slight and should be carefully searched for.
c. *Bleeding* – Scant to moderate (less than 500ml) dark vaginal bleeding is present.
d. *Mother* – Vital signs remain unchanged.
e. *Foetus* – Foetal heart sounds are strong and regular.

Moderate Antepartum Haemorrhage:

Mild separation may progress to a moderate separation, or, the onset of symptoms of moderate separation may be abrupt.

a. *Pain* – There is a continuous abdominal (uterine) pain which may be severe.
b. *Bleeding* – Although only moderate vaginal bleeding may occur the total blood loss may be over 1 litre.
c. *Tenderness* – Uterine tenderness is clearly present. Sometimes the tenderness is generalised and there is muscle guarding with rebound tenderness. The tenderness is due to uterine infiltration with blood.
d. *Mother* – The patient may be shocked, with hypotension, tachycardia and a cold, moist skin, but often has hypertension, associated with reduced renal output and proteinuria.
e. *Foetus* – The foetus may show signs of foetal distress and succumb.
f. *Pre-eclampsia* – May occur here but is more prominent with severe separation.

Severe Antepartum Haemorrhage:

Here the onset is usually abrupt.

a. *Pain* – Uterine pain is agonizing.
b. *Tenderness* – The uterus is tender, tense, and "woody hard"; probably due to a reflex tetanic contraction.
c. *Bleeding* – External bleeding may be moderate or *absent*

(concealed haemorrhage). *At least* 2 litres of blood have been lost from circulation.

d. *Mother* – The patient is usually hypertensive initially but quickly becomes shocked. (The degree of shock cannot be related to the external blood loss.)
e. *Foetus* – The foetus almost invariably dies.
f. *Pre-eclampsia* – This is present in about one third of cases giving rise to proteinuria, and raising the blood pressure to almost normal level in spite of shock.

Complications:

a. *Hypofibrinogenaemia* is an uncommon but extremely dangerous complication of acidental haemorrhage, and can occur after any degree of abruption, but more commonly after the severe concealed variety.

The patient has an attack of abdominal pain, following placental separation, and the typical tense, tender woody uterus occurs. After an interval of time bleeding is seen, and despite adequate therapy, blood transfusion and delivery of the infant, the bleeding continues.

If a sample of blood is taken and examined, it is seen that either no clot forms or, if a clot does develop, it can be easily broken up by gentle shaking of the test tube. This condition is known as hypofibrinogenaemia, and the patient may bleed to death unless adequate therapy is instituted to correct the abnormality.

How Does Hypofibrinogenaemia Occur?

There are thought to be two major mechanisms involved which may act separately or together to deplete fibringogen levels.

i. Following the haemorrhage there is a release of thromboplastins into the blood stream from placental tissue. This thromboplastin causes a generalised intravascular deposition of fibrin which, although not sufficient to block the vessels, causes a laying down of a thin thrombus on the vessel walls. This fibrin deposition causes a reduction in fibrinogen levels to below 0.1 gm %. As the fibrinogen level falls, the blood ceases to coagulate, and uncontrollable haemorrhage may occur.

ii. Fibrinolytic system: Normal plasma contains a complicated fibrinolytic system which aids in the control of the haemostatic mechanism. This system consists of pro-activators, an inactive precursor (plasminogen), an active enzyme (plasmin), and various inhibitory substances. The pro-activators are released from placental tissues. Plasmin is able to hydrolyse fibrinogen and other clotting factors and destroy their effectiveness. Fibrin degradation products then act to inhibit thromboplastin conversion of prothrombin to thrombin.

The effectiveness of the conversion of plasminogen to plasmin can be prevented by competition by a certain amino-acid known as epsilon amino caproic acid (EACA). These amino-acids act to inhibit the activators of the plasminogen.

b. *Renal Failure* may occur due to the resultant renal ischaemia which develops following hypovolaemia and arteriolar constriction. It is important in the prevention of renal failure to resuscitate the patient adequately, preferably with whole blood.

c. The uterus may have multiple bruising or *ecyhmoses* on its serosal surface due to the blood which tracks through muscle and parametrial tissue (Couvellaire uterus).

Management of Accidental Haemorrhage:

The initial management can be divided into two types, depending on the severity of the condition.

a. *Mild, Revealed Accidental Haemorrhage*
In this case there is often a small amount of bleeding associated with minimal pain and tenderness. The condition may occur before the 36th week, so it is essential to gain foetal maturity whenever posssible.

i. Put the patient to bed in hospital.
ii. Analgesic – morphia 15 mg or pethidine 100 mg.
iii. Cross match blood. Estimate Hb and haematocrit.
iv. Have fibrinogen available if necessary.
v. Observe patient for several days until bleeding and pain subside, then allow out of bed, after excluding a placenta praevia. If no other contra-indications are present, the patient may be allowed home. These patients must not be allowed to go postmature, because the placental function has already been reduced due to the accidental haemorrhage.

b. *Moderate/Severe Accidental Haemorrhage*

These cases are suffering from severe pain, are bleeding, and have lost more blood than is usually estimated. They may go into irreversible shock, suffer renal damage or hypofibrinogenaemia unless treated adqeuately.

i. Put to rest in bed in hospital.
ii. Give morphia 15mg.
iii. Cross match and begin transfusing blood. The patient invariably loses twice as much as is estimated. To prevent the patient suffering shock a drip must be set up as early as possible.
iv. Check the blood from a vein to determine fibrinogen levels. A quick test can be performed by placing blood in a test tube and inverting every 30 sec. Normally the the blood will clot and remain a firm matrix within 1-2 minutes. Any delay in clotting necessitates a full fibrinogen estimation, but do not delay treatment. Give fibrinogen 2, 4 or 6 gm. as required, or fresh whole blood.
If it is suspected that fibrinolysins are responsible for the coagulation defect and these factors are demonstrated as absent, then EACA can be given, 4-6 gm initially then 1 gm/hour.
v. When the patient is resuscitated, rupture the membranes to precipitate labour. Often, however, one will find the patient is already in labour following the initial tetanic contraction, but if not an oxytocin drip may be set up.
vi. Deliver the infant as quickly as necessary, but take special care that post-partum haemorrhage does not take place. Ergometrine must be given following delivery to gain maximum amount of advantage from uterine contractions.

Mortality:

The mortality rates depend entirely on the degree of severity of the condition.

The maternal mortality is in the vicinity of 7% for severe types of accidental haemorrhage, mainly from associated hypofibrinogenaemia and renal failures.

Foetal loss is in the vicinity of 95% for severe concealed accidental haemorrhage and drops to about 5-10% for cases of mild degree.

There is an overall foetal loss of about 25% in all cases of accidental haemorrhage.

Differential Diagnosis:

In mild cases differentiation from a placenta praevia may be difficult. Rectal and urinary bleeding as a cause of haemorrhage must always be excluded.

A diagnosis of accidental haemorrhage can only be made in the presence of pain and tenderness of the uterus, whereas the other types of A.P.H. are usually painless.

A concealed accidental haemorrhage may be confused with appendicitis, urinary tract infection, other causes of intra-abdominal bleeding, torsion of a viscus, or rupture of the uterus. A careful history is essential together with complete physical and laboratory examination.

3. **Lesions of the Cervix, Vagina, and Vulva**

This is an uncommon cause of bleeding in late pregnancy. A detailed history and carefully examination with a Sim's speculum will help locate the site of bleeding. Treatment of these conditions is discussed in the appropriate chapters. The lesions may be cervical carcinoma, cervical erosion or polyps, varicosities, vaginitis, or trauma.

4. **Vasa Praevia**

In this rare condition there is a velamentous insertion of the cord in association with a placenta praevia, so that foetal cord vessels pass across the cervical opening. Bleeding here is of *foetal* origin.

General Management of a Patient with Antepartum Haemorrhage:

The assessment of a patient who has been bleeding in late pregnancy begins with adequate *history* (if the condition permits). One must exclude placenta praevia and abruptio placentae, therefore relevant questions concerning the respective symptoms are asked.

In the *examination* such points are looked for as blood pressure uterine tenderness, presence of the foetal heart sound, and the source of bleeding on speculum examination.

The patient is admitted to the ward and rested in bed until special investigations have made the diagnosis more clear. More definitive treatment may then be commenced, if it is indicated.

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CHAPTER 8

FOETAL WELL BEING

General Instructional Objective

Recognises parameters of and understands factors affecting foetal well being so that appropriate management can be instituted.

Specific Behaviours

1. Describes factors which may impair foetal well being.
2. Distinguishes "at risk" from normal pregnancies.
3. Demonstrates ability to assess foetal well being.
4. Explains the importance of antenatal care on foetal well being.
5. Evaluates screening and diagnostic procedures used during pregnancy to assess foetal well being.
6. Discusses the management of the "at risk" pregnancy.

Foetal Well Being

During a pregnancy the normal development and maturation of a foetus may be influenced by a variety of factors many of which are known and most of which are either preventable or treatable. During labour the survival of the foetus may be threatened by hypoxia or biochemical or nutritional failure and this may be manifest by signs of foetal distress. The obstetrician's task, therefore, is the early recognition of the foetus at risk and the provison of the best possible

management of the problem, both during the pregnancy and the labour.

A. Factors which may impair foetal well being during pregnancy. (The high-risk pregnancies)

The known factors may be classified as being maternal, placental, or foetal.

1. **Maternal Factors:**

   a. *Hypertension* (any cause)
   Placental vascular lesions such as acute fibrinoid necrosis, hyalinisation, and possibly arteriolar spasm may lead to infarction and *placental insufficiency.* (Chapter 5).

   b. *Urinary Tract Infection*

   Bacterial toxins are thought to produce fibrinoid changes within vessels reducing blood flow. This is followed by increased incidence of antepartum haemorrhage, premature labour, intrauterine death, and neonatal death. (Chapter 11).

   c. *Diabetes Mellitus*

   Several factors are important here, including an increased incidence of urinary tract infections and pre-eclampsia, maternal ketosis, and intrauterine death of obscure origin. (Chapter 11).

   d. *Anaemia*

   Anaemia is associated with increased rate of prematurity and low birth weight (Chapter 11).

   e. *Elderly Primigravida*

   In primigravidae over the age of *35* stillbirths are more common, predominantly due to placental insufficiency or accidental haemorrhage (see page 167 for further discussion).

   f. *Rh Isoimmunization*

   Foetal red blood cell destruction by maternal antibodies may progress to anaemia, cardiac failure, and generalised oedema or hydrops foetalis. (see page 160 for further discussion).

   g. *Other Factors Include:*

      i. Multiple pregnancy – reduced placental area.
      ii. Nutritional factors – smaller babies.
      iii. Low socio-economic status – smaller babies.
      iv. Smoking – smaller babies.
      v. Grand multipara – more complications (see page 168 for further discussion).
      vi. Poor obstetric history – increased risk of problems.
      vii. Maternal syphilis – intrauterine death (Chapter 13).
      viii. Uterine malformations and fibroids.

2. **Placental Factors:**

   a. *Antepartum Haemorrhage*

   The extent of placental separation determines foetal nutrition and future foetal growth capacity. (Chapter 7).

   b. *Prolonged Pregnancy*

   Placental efficiency declines after term with reduction of blood flow and, therefore, of transplacental nutrient transfer.

   c. *Other Placental Factors*

   Infarcts, haemangiomata, single artery – associated with reduced placental efficiency. Cord about foetal neck – may cause reduced blood flow.

3. **High-Risk Pregnancy Identifications:**

   a. Early detection of the foetus at risk depends on the patient's early and regular attendance, and the obstetrician's clinical skill, in the antenatal clinic.

   An accurate initial *history* will help detect many of the abovementioned factors such as urinary tract infection, diabetes, the elderly primigravida, smoking, poor obstetric history and others.

   The *examination* will confirm some of the above and unmask others such as hypertension. Routine and special *investigations* will help diagnose anaemia, Rh status, syphilis, rubella, or diabetes mellitus.

   b. Detection of some conditions can only be made during the various stages of pregnancy. In this respect regular maternal

attendance at the *antenatal clinic* is of paramount importance to the foetus. Pre-eclampsia, urinary tract infection, anaemia, Rh isoimmunization, and antepartum haemorrhage must actively be searched for.

c. *Monitoring of Foetal Growth and Well-being* may be carried out during the latter part of pregnancy by the use of the following methods:

i. *Ultrasonic Echoscopy* – accurate measurement of the bi-parietal diameter of the foetus can give a growth rate curve when performed every 7 to 10 days. This may be started at 28 weeks to establish an early baseline.

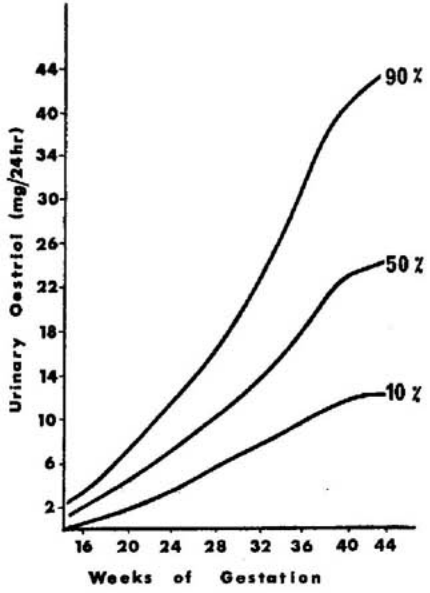

Fig. 8.1. Twenty-four hour urinary oestriol. (After Lundy *et al*, 1973).

ii. *Urinary Oestriol Levels* – The integrity of the feto-placental unit may be assessed by urinary oestriol excretion. However, this method requires intelligent interpretation since daily variation can be of the order of 30-50%, making a single estimation virtually useless. Using *daily* estimations a *trend* is established. The normal trend is a rising one, a falling trend may indicate decreasing foetal or placental function. Normal lower limits (Fig. 8.1) are not absolute but may be taken as the 10th percentile of the distribution.

Oestriol assays are carried out on 24 hour urine samples and may be commenced, for an at-risk foetus, at about week 30, since little can be done for the foetus if a falling trend is seen prior to this time.

iii. *Lecithin/Sphingomyelin Ratio* – Since the alveolar surfactant required for normal ventilation is made up largely of lecithin, the amniotic concentrations of the above phospholipids can be used to predict foetal respiratory maturity. In normal pregnancy an increased production of lecithin in relation to the production of sphingomyelin at about week 34 (Fig. 8.2) is associated with lung maturation. Delivery of a foetus after this time correclates closely with markedly reduced occurrence of respiratory distress syndrome.

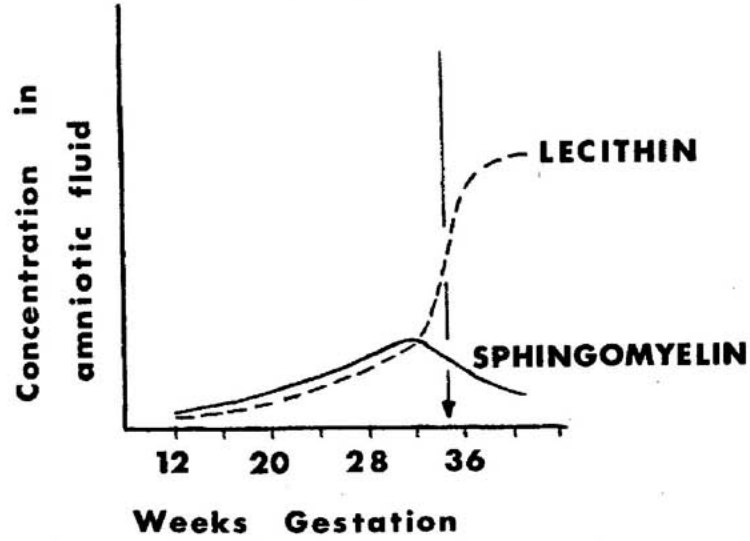

Fig. 8.2. Amniotic lecithin and sphingomyelin concentrations in normal pregnancy. The 1:1 ration of early pregnancy reaches a value of 2:1 or more at about 34 weeks gestation. The increased L/S ration is an index of foetal lung maturity. (From Gluck *et al*, 1973).

In the high-risk pregnancy maturation of the foetal lung is known to be accelerated by maternal hypertension, antepartum haemorrhage, and prolonged rupture of membranes. The foetuses of some diabetics have delayed

lung maturation. (Gluck *et el*, 1973). Corticosteroids may be the mediators of the accelerated maturation and thus their use may prove useful in the high-risk patients prior to delivery.

iv. *Amniotic Bilirubin* – Intrauterine foetal haemolysis leads to a spill-over of bilirubin into the amniotic fluid. Measurement of its concentration is of prognostic value and is used as a guide in management (see page 160 for further discussion) of cases of rhesus incompatibility.

v. *Radiology* – Estimation of foetal maturity is by no means accurate and is of little value prior to the calcification of the distal femoral (36 weeks) or the proximal tibial epiphyses (38 weeks).

d. *Clinical Measurements* – A crude clinical estimation of foetal growth can be made by measuring the maternal girth and fundal height. From weeks 28 to 36 the greatest abdominal circumstances is taken to increase at a rate of 1"/week being 36 inches at 36 weeks. Fundal height, measured from the symphysis pubis, rises at a rate of 1cm/week, reaching 36 cm at 36 weeks.

B. Antenatal Care

The objects of antenatal care are:

1. To promote and maintain good physical and mental health of the mother during pregnancy.
2. To ensure a mature, live, healthy child.
3. To prepare the mother for labour, lactation and the subsequent care of her child, from the physical, psychological and domestic point of view.
4. To detect at an early stage any medical or obstetrical abnormality which might endanger the life or impair the health of the mother and baby and treat this abnormality appropriately.

When meticulous attention is paid to all aspects of antenatal care by the obstetrician maternal and foetal morbidity and mortality is significantly reduced. This will not be possible, however, if maternal attendance is not regular. The explanation of these facts to the patient is the obstetrician's duty.

With the "captive" population of women who attend the clinic any

disease may be screened for and detected early. Such screening will remove foetal wastage due to diseases such as rubella, venereal disease, or isoimmunisation. Regular attendance allows also for the care of the mother and the foetus considered to be suffering from those "high-risk' maternal conditions which may lead to foetal death.

Maternal education through counselling, lectures, discussion groups, and films concerning labour, the puerperium, and motherhood, will prepare the patient psychologically for those future events. Misconceptions and problems can be aired at the clinic, and this will help to resolve much unnecessary anxiety.

C. Foetal Distress

The term 'foetal distress' is usually applied to the acute situation occuring during labour when such signs as foetal tachycardia (greater than 160/minute), bradycardia (less than 100/minute), or meconium staining of liquor amnii indicate the foetus may be deprived of elementary nutrition. This is usually precipitated by biochemical hypoxia and hypoglycaemia. Twenty per cent of babies showing signs of foetal distress will also have a blood pH of less than 7.15.

Some 10%-15% of patients who go into labour can be labelled as 'high risk' pregnancies. Due to their chronic nutritional deprivation resulting in reduced glycogen stores many of the foetuses in these pregnancies will be unable to withstand the rigors of a difficult or prolonged labour and thus will be more prone to show signs of foetal distress during labour.

Statistically, of 1000 mothers going into labour 140 of their foetuses will have signs of foetal distress, either as sustained bradycardia, meconium-stained liquor, or both. Of these, only 40 foetuses will have a low blood pH, and of this 40, 20 are at risk of death unless active measures are taken. This is only 2% of the original 1000 mothers. To perform Caesarean section with it's 7-fold increase in maternal mortality on the original 14%, because signs of foetal distress were present, would result in greater morbidity and mortality than if only the 2% were to be considered for Caesarean section. Note that of the abovementioned 140 mothers, 80 will come from the 'high-risk' pregnancy group previously mentioned.

There is, therefore, a need for a method of deciding which babies are at risk, and consequently which babies will require to be delivered before the normal end of the labour.

Two groups of babies can be dealt with fairly easily. In those cases

in the second stage of labour with the head engaged in the pelvis, there is no need for differentiation and the baby should be delivered by forceps. In those high-risk cases at the beginning of labour with marked signs of foetal distress and an unfavourable obstetrical situation (high presenting part, weak pains and tight cervix), the decision for abdominal delivery is not a difficult one.

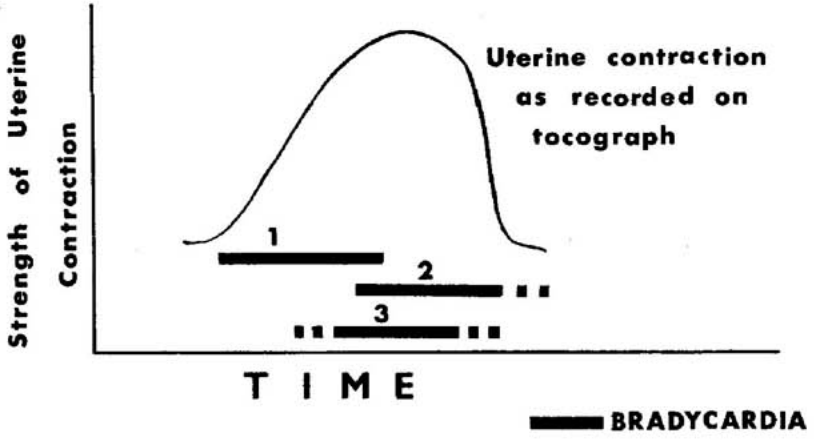

Fig. 8.3. Patterns of foetal bradycardia expressed in relation to uterine contraction. 1. Early or head compression.
2. Late or utero-placental compression.
3. Variable or cord compression.

But the large bulk of cases manifesting signs of foetal distress lie between the above two extremes, and are worrying clinical problems. On finding foetal distress during labour the cause and severity of the distress must be determined. Continuous foetal heart monitoring combined with monitoring of uterine activity by tocography may show three patterns of bradycardia (Fig. 8.3).

These are:

1. **Early, or Head Compression** characterized by –

   . early onset,
   . bradycardia usually of not less than 100/min,
   . bradycardia usually shorter than 90 seconds duration,
   . not affected by breathing oxygen.

2. **Late or Utero-Placental Compression**

   . comes on late in the uterine contraction,

   . lasts a variable time after the end of the contraction.

3. **Variable or Cord Compression**

   . variable onset and foetal heart rate,
   . bradycardia usually falls below 100/min, baseline being normal,
   . duration of bradycardia varies from 10 sec to minutes,
   . markedly altered by maternal position change.

In the high-risk pregnancy any sustained bradycardia of less than 100/min, should be followed by a determination of foetal blood pH. A persistent pH of less than 7.15 is associated with Apgar scores of less than 6, and in these cases, therefore, immediate delivery by Caresarean section or forceps, is indicated. Since selection criteria are not yet accurate, many operative procedures are probably unnecessary.

Management of the High-Risk Pregnancy:

Each condition that creates a high-risk pregnacy has its specific management and is discussed in detail elsewhere. The *general* management of a high risk pregnancy, however, will involve the following major components:–

a. Early diagnosis antenatally.

b. Alteration of the antenatal routine to suit the particular condition. For example, the patient with diabetes mellitus is admitted to hospital at the first visit for stabilisation, and is then seen at least every 2 weeks and not the usual 4 weeks. The patient with a multiple pregnancy will require extra iron and folate supplements and will be brought into hospital for rest at 30 weeks to reduce the likelihood of premature labour.

c. Alteration of the delivery procedure. In some cases time of delivery will be dictated by the oestriol levels, echoscope findings, and lecithin/sphingomyelin ratios. Most diabetic patients will have labour induced at 37 weeks. The time of delivery, and the method, again depends on the particular condition.

D. Induction of Labour

Induction of labour is an attempt to terminate a pregnancy artificially at any time after 20 weeks gestation, aiming at a vaginal delivery. The incidence of induction varies between 3 and 25%, depending on

the policy at various hospitals. At the Royal Hospital for Women it is approximately 20%.

In general terms, a pregnancy should be terminated if it's continuation endangers the life of the mother, or if it is felt that the foetus is at a greater risk in the uterus than if it were in a crib. The indications for induction will be listed.

Maternal Indications for Induction of Labour

Eclampsia
Pre-eclampsia
Hypertension
Chronic pyelonephritis
Accidental haemorrhage
Placenta praevia
Intrauterine death

Foetal Indications for Induction of Labour

Rhesus iso-immunisation
Diabetes mellitus
Recurrent intrauterine death
Placental insufficiency
Prolonged pregnancy

Methods of Induction:

Labour may be induced medically or surgically.

a. *Medical Induction*

i. Oil, enema, and shower – of doubtful efficiency.
ii. Quinine sulphate – not very effective and seldom used.
iii. Hormones – a) Oestrogen – may be used in cases of intrauterine death but is not regarded as being of any marked value.
b) Prostaglandins – may be used successfully to induce labour but intense nausea and cardio-vascular reactions have reduced its use and it is not generally advocated.
c) Oxytocins – oxytocin stimulates uterine contractility, but in larger amounts has an anti-diuretic action. A synthetic preparation, Syntocinon (Sandoz), is now commonly used.

Oxytocin may be administered as:

. Intravenous infusion
. Buccal Pitocin (tablets)
. Nasal Pitocin (spray)
. Nasal Syntocinon (spray)

Intra-muscular Syntocinion should never be used prior to delivery because of the danger of uterine rupture.

Oxytocin may be used as the only method of induction, or (associated with) artificial rupture of the membranes, in which case the success rate will be over 90%.

Close supervision is required when using an oxytocin infusion because the patient's sensitivity to the drug is not known. She may respond to it by a tetanic uterine contraction causing foetal hypoxia, and occasionally rupture of the uterus may occur.

Contraindications to Oxytocin Infusuion include:

1. *Grand Multiparity* – This is not an absolute contra-indication but great care is necessary, since risk of uterine rupture is high.
2. *Previous Caesarean Section* – This is another relative contraindication, since the chance of scar rupture is high.
3. *Absolute Disproportion*
4. *Incoordinate Uterine Activity* – These cases are unlikely to profit from the oxytocin while the risk of uterine rupture is increased.

b. *Surgical Induction*

Amniotomy is the usual and most efficient method for induction of labour. Either a high or low artificial rupture of the membranes (L.A.R.M.) can be performed.

Hindwater (high) rupture is less likely to be followed by cord proplapse or infection than forewater (low) rupture. Its chief place is in drainage of excess liquor if the presenting part is high (in cases of polyhyhramnios when the head becomes well applied to the cervix, a L.A.R.M. should follow). The main factor affecting success of amniotomy is the state of the cervix. "Success" means that labour ensures within 24 hours of amniotomy. If the

cervix is "ripe" admitting two fingers, soft and effacing – induction will succeed in 95% of cases. With a long, firm cervix admitting only one finger the success rate falls to 65%.

Risk of Induction of Labour:

1. **Failure**

   If the patient is not in labour 48 to 72 hours after induction, Caesarean section is almost inevitable. The main hazard to both mother and baby is sepsis, and anitibiotics should be administered within 24 hours to reduce its incidence (see Chapter 4).

2. **Prematurity**

   This risk must always be borne in mind, especially when there is some doubt as to the date of the L.M.P. and the time of conception.

3. **Disordered Uterine Action** may result when induction with oxytocin is attempted in a uterus which is not ready (see Chapter 4).

4. **Complications of Surgical Induction.**

   a. *Sepsis* – If the patient is not delivered by 24 hours from the time the membranes were ruptured the incidence of infection rises rapidly. The incidence of uterine infection doubles for every 6 hours that elapses after 24 hours of ruptured membranes. For this reason, penicillin and streptomycin are usually ordered when the membranes have been ruptured for 24 hours.

   b. *Cord Prolapse* – with the engaged head the incidence is 0.1% but rises to 2-5% with a high head. The risk is not completely abolished with high rupture, as the forewaters also may be inadvertently ruptured.

   c. *Accidental Haemorrhage* may follow the sudden release of a large volume of liquor in cases of polyhdramnios, in which a slow release should be made or, alternatively, abdominal paracentesis carried out.

   d. *Amniotic Fluid Embolism* is rare but often lethal. Liquor, containing debris, meconium, and hair, may enter a maternal sinus, usually during or after a high rupture and is often associated with maternal death or severe hypofibrinogenaemia.

Suggested Method of Induction:

a. Empty the lower bowel. This will remove obstruction to the descent of the presenting part.

b. *Amniotomy* with strict aseptic precautions is carried out next, provided that the foetal head is well applied to the cervix. A careful vaginal examination is made prior to the amniotomy, to check pelvic dimensions, state of the cervix, and position and nature of the presenting part. The index or both index and middle fingers are then passed through the cervix and the membranes stripped widely from the lower uterine segment. Then the left hand guides a pair of artery forceps along the fingers of the right hand. The membranes are grasped, tented and ruptured by pulling on the forceps. The hole made in the membranes in enlarged by the fingers of the right hand which remain in the vagina, controlling the escape of liquor amnii until the presenting part is firmly settled in the pelvic.

c. The patient is observed at regular intervals for the onset of uterine contractions and for evidence of foetal distress.

d. *Oxytocin Drip.* If the indication for induction is very urgent an oxytocic drip can be set up at the time the membranes are ruptured. But if this is not absolutely necessary it should be withheld as 80% or more of the patients who have their membranes ruptured will proceed with a normal labour. If labour has not ensued 12 hours after amniotomy an oxytocic drip should be commenced, with antibiotics soon after. At the Royal Hospital for Women 10% to 15% of women who are induced require an oxytocin drip.

E. Intrauterine Death

Foetal death before 20 weeks results in an abortion.
Infrequently foetal retention will result in a missed abortion (see Chapter 6).
After the 20th week intrauterine death may be diagnosed by the following *signs and symptoms:*

- Maternal lack of 'pregnant feeling' with regression of breast changes.
- Prolonged absence of foetal movements.
- Lack of foetal heart sounds on auscultation and with the Doptone Detector.

. Cessation of uterine enlargement.
. Falling or very low oestriol levels in the urine. (Pregnancy test may remain positive for up to a month.)
. A collapsed foetal skull on palpation, giving a "grating" feeling as the skull bones override each other.
. X-ray changes include:

i. *Overlapping of skull bones* (Spalding's sign) which take about 3-4 days to develop, and is due to reduction of intracranial contents and softening of ligaments.

ii. *Increased flexion of the spine* (Ball's sign) which takes at least 2 weeks to develope.

iii. *Gas in the great vessels*, usually present about 36 hours after death.

Management:

During the 2 to 3 week period after foetal death in which the diagnosis is being made, 70 to 90% of dead foetuses are expelled. At this stage psychological support for the patient is of paramount importance. If delivery does not occur within 3 weeks, induction of labour should be undertaken in anticipation of the development of hypofibrinogenaemia. A syntocinon infusion may be used for induction. If some viable placenta still produces progesterone which reduces uterine muscle excitability however, oestrogens may be used to block this action.

It is wise not to rupture membranes in the presence of a dead foetus as labour may not follow and the risk of infection is high.

F. Isoimmunisation

1. Rhesus (Rh) Factor

The "Rh factor" was first detected in 1940 by Landsteinder and Wiener but it was not till 1944 that Fisher discovered the individual antigens that made up the "Rh factor". There are 6 main Rhesus antigens (three pairs) which are inherited by 6 separate genes, C, c, D, d, E, e. Three genes (one from each pair) are located on each of a pair of chromosomes, one choromosome being inherited from each parent. It is the presence or absence of the D antigen, which alone determines if a person is Rh positive or Rh negative.

In European races 83% of people are Rh positive, 50 of whom are heterozygous and 33 homozygous for the D antigen.

In pure Asian and Polynesian races there are no D negative individuals, very few in Indians and only 0.3% in Japanese.

Rhesus isoimmunisation may occur when a Rh-negative mother is pregnant with a Rh positive foetus. The foetal cells may leak into the maternal circulation (foeto-maternal haemorrhage) and as they contain blood group proteins (antigens) absent in the mother, maternal antibody formation may be stimulated.

Maternal sensitization is more likely to occur after such procedures as induced abortion, external version (especially if performed under anaesthesia), difficult instrumental deliveries, manual removal of the placenta, accidental haemorrhage and Caesarean Section.

It may also occur at the time of placental separation in the 3rd stage of labour, during amniocentesis, and after transfusion with Rh+ve blood. Maternal antibody formation depends on the size of the foeto-maternal haemorrhage, the time at which it occurs, the individual sensitiviety of the mother, and on the ABO blood groups. If the baby's ABO grouping is incompatible with the mother, then the mother's anti A and/or anti B antibodies rapidly destroy the foetal red cells, and maternal sensitization is much less frequent. An Rh–ve woman may marry an Rh–ve husband and his children will all be Rh–ve. With a heterozygous D husband only Rh+ve offspring are at risk. It is rare for the first Rhesus positive pregnancy to sensitize a woman, but 1 in 10 women are sensitised by two such pregnancies. This risk is doubled (1/5) if both Rh+ve pregnancies are ABO compatible with the mother. At present about 6/1000 babies are affected in this way, one of which will develop a sufficient degree of anaemia to produce a stillbirth. In the first pregnancy in which Rh antibodies occur the risk of stillbirth is about 10%. Foetal red blood cells can be detected in the maternal blood by the Kleihauer count, which is based on the fact that foetal cells contain Hb-F in contrast to the Hb-A of adult haemoglobin. When treated with a pH 3.3 buffer, foetal cells stain in much the same way as usual, while maternal cells lose their haemoglobin and appear as "ghost" cells. Following sensitization two types of antibodies develop in the mother:

a. Complete (Saline) or IgM antibodies. These are detected first and do not cross the placental barrier because of their size (about 890,000 mol. wt.).

b. Incomplete (Albumin) or IgG antibodies. Being much smaller

(mol. wt. 140,000) they readily cross the placenta resulting in varying degrees of destruction of foetal red blood cells.

2. **Foetal Response**

Foetal haemopoeitic tissues attempt to compensate by increasing the rate of red cell formation, as evidenced by a reticulocyte count of over 6%. The liver, spleen and placenta are enlarged by areas of haemopoeisis.

The foetal red blood cell destruction leads to an increase in bilirubin production, most of which is removed into the maternal circulation by the placenta. Some bilirubin enters the liquor amnii and its concentration is used to determine the severity of the haemolysis. Kernicterus, therefore, will not develop in utero.

Following delivery jaundice may appear within 48 hours in mild cases, and within 12 hours in severe cases due to the rapid rise in bilirubin, unless this is prevented by multiple exchange transfusions. If the bilirubin level rises above 20 mg% there is an increased risk of developing kernicterus, especially in the premature infant.

With severe intrauterine anaemia due to haemolysis, the foetus will develop cardiac failure with generalised oedema, known as hydrops foetalis, followed by intrauterine death.

3. **Management**

Prevention of Sensitization

It is now possible to eradicate foetal wastage due to Rh immunisation. This requires expert care of all Rh-ve women during *all* stages of their reproducitve lives.

Even before a pregnancy occurs all transfusions should be done using Rh-ve blood for the Rh-ve woman.

Early in the pregnancy blood grouping and antibody check is carried out. In the unsensitized patient with such conditions as an abortion (after 8 weeks) or anterpatum haemorrhage, human Anti-D gamma globulin is given intramuscularly in the dose of 200 mg (1ml.) unless it is known with certainty that the husband is Rh–ve and the child is his. Apart from the above conditions, the D antigen will not immunise 98% of women during the pregnancy – for an, as yet, unknown reason.

The test for Rh antibodies is repeated at 26-28 weeks, and if found negative, the pregnancy is continued as a normal one. At 28 and 34 weeks anti D gamma globulin is given to the unsensitized Rh-ve woman to mop up any cells that may leak into the maternal circulation before delivery, as occurs in a small number of patients. In the management of the third stage of labour care should be taken to minimise the risk of foeto-maternal transfusions.

The administration of oxytocics should be withheld until the clamp is removed from the placental end of the cord. Cord blood is collected into a plain tube and an oxalated tube, and is sent to the laboratory for blood group determination. If the baby is Rh+ve immunisation may have occurred and a direct Coomb's test is requested to determine whether foetal red blood cells are coated with incomplete antibody (Fig. 8.4). If the test is negative, the cells being not coated, anti D gamma globulin is given to the mother *within 72 hours of delivery*. If the Coombs test is positive, then order –

a. Cord haemoglobin, and
b. Cord plasma bilirubin,

to assess the severity of the haemolysis.

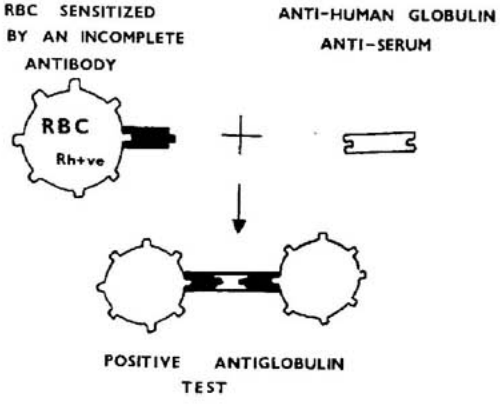

Fig. 8.4. The Direct Coomb's Test. The foetal red blood cells carrying Rh +ve antigens are coated with maternal incomplete antibodies. Addition of anti-human globulin antiserum leads to an agglutination of these cells giving a positive result. This indicates both the presence of maternal iso-immunisation and potential foetal haemolysis.

Management of the Sensitized Pregnancy:

All Rh-ve women should be tested for the presence of antibodies

the first time they are seen in pregnancy, and the test repeated at 26 to 28 weeks. If anti-D antibodies are detected (titre is not quantitative here, and is unimportant) at any stage, amniocentesis at *28 weeks* is performed and repeated as often as indicated. If there is a previous history of severe haemolytic disease the first amniocentesis may have to be done much earlier. perhaps even at 20 weeks gestation. The method of amniocentesis is illustrated in Fig. 8.5. The amniotic fluid is protected from sunlight during its transport to the laboratory, as the light may lower the bilirubin concentration by transforming it to a colourless compound. The laboratory will determine the size of the pigment peak in terms of optical density at 450 mμ. (Fig. 8.6).

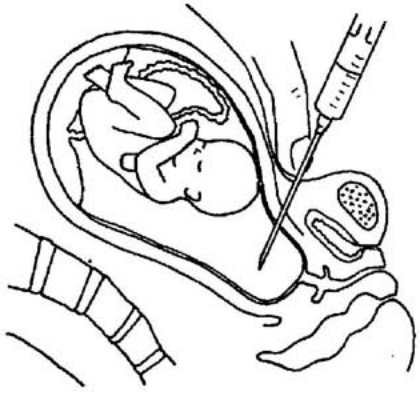

Fig. 8.5. Amniocentesis. After emptying the baldder the skin is prepared and the foetal presenting part lifted away from the symphysis pubis. A 7.5 cm. 21 gauge needle with an attached syringe is inserted in the midline, an inch below the presenting part (From Preda, 1973).

Because the bilirubin level normally falls near term the amniocenteis result is plotted on a graph (Fig. 8.7) to show the severity of the condition and to assess whether transfusion is required. At 28 weeks, for example, an optical density of more than 0.2 will indicate a need for an intrauterine blood transfusion, in which *Rh-ve* packed cells are injected slowly into the foetal peritoneal cavity. Multiple transfusions may be required.

Depending on the result of the first amniocentesis, a second one is carried out up to 4 weeks later. This result will dictate either conservative management or a need for transfusion.

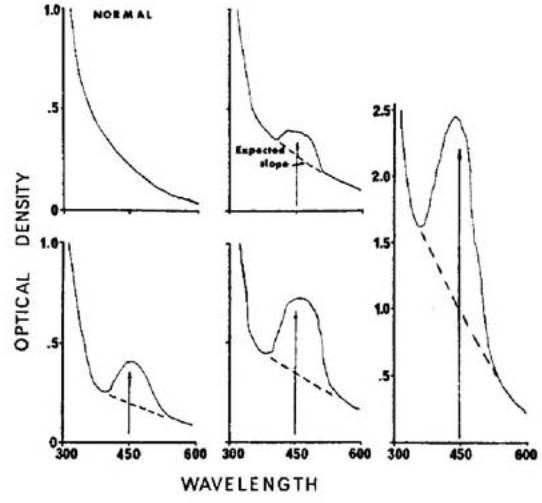

Fig. 8.6. Serial tracings of amniotic fluid specimens demonstrating the progressive development of the abnormal curve (From Freda, 1973).

No foetus suffering from hemolytic disease should be delivered later than 38 weeks amenorrhoea and no anaemic baby should be delivered before 34 weeks (as severe anaemia and gross prematurity are not compatible with foetal survival). In these cases it is important that the *duration of gestation* and not the period of amenorrhoea is determined accurately. If maturity is uncertain it is better to wait.

When the time for delivery has been decided on, induction of labour by L.A.R.M. and oxytocin infusion in a well-equipped hospital is carried out.

The management of third stage of labour in these cases is the same as that described for the unsensitized mothers.

G. Prolonged Pregnancy

Definition:

Pregnancy is considered to be prolonged when it continues 2 weeks past the expected date of confinement, i.e. 297 days (42 weeks and 3 days) past the first day of the L.M.P. Some 5% of all pregnancies go beyond 297 days.

The baby tends to be slightly heavier than normal, with a dry skin, long nails and little vernix caseosa. The foetal head is firm, the skull

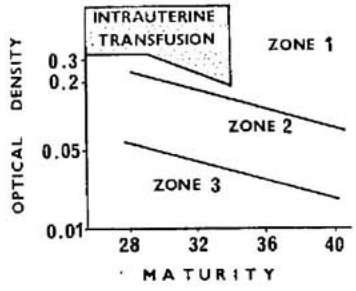

Fig. 8.7. Chart used for assessing foetal prognosis in Rh haemolytic disease. Amniocentesis results may fall in one of three zones. In Zone 1 the baby is mildly affected and can probably be safely allowed to go to 38 weeks gestation. In Zone 2, a somewhat unreliable zone, multiple amniocenteses may indicate in which direction the trend is developing. Earlier induction will be required. In Zone 3 after 34 weeks immediate delivery should be undertaken. Prior to about 34 weeks intrauterine transfusions should be carried out. Note that after this time transfusions need not be carried out since a transfusion at 33 weeks will suffice the baby for at least two weeks in which time it will be delivered. (From Bosch *et al*, 1974).

bones are well ossified and do not mould readily. There is often oligohydramnios.

Significance:

The rate of foetal distress is doubled at 297 days as compared with 283 days amenorrhoea, and stillbirth and neonatal death rate increases significantly. Dystocia is more common.

Causes of Perinatal Deaths:

The majority of deaths are intrauterine. *Placental insufficiency* is a major cause. Evidence of lowered oxygen in the umbilical vein has been found but this is not universal and controversy exists as to the exact cause of foetal distress. It is true that placental infarction occurs, but this is not matched by the fall in efficiency.

The biparietal and occipito-frontal diameters of the skull increase by one millimetre/week after 36 weeks of pregnancy. In addition, the bones become more ossified, harder, and less easily moulded as pregnancy continues past term. Sometimes, a high foetal head due to an abnormally shaped pelvis, is a poor stimulus to labour. These causes contribute to the increased incidence of dystocia.

Diagnosis:

The criteria for accurate diagnosis are:—

1. Known dates.
2. Normal last menstrual period – and not an implantation bleed.
3. Regular menstrual cycle.
4. Early confirmation of uterine size. An examination in the first 3 or 4 months of pregnancy will confirm that the uterine size corresponds to the dates.
5. Onset of foetal movements confirms the estimation of dates.
6. Radiology of distal femoral and proximal tibial epiphyses may be useful. These are usually visible at 36 and 38 weeks respectively.
7. Volume of liquor amnii – diminishes beyond term, and may be estimated by uterine palpation, maternal girth, or a fall in maternal weight.
8. Oestriol excretion may show a falling trend.

Management:

No patient with hypertension, pre-eclampsia, Rh-isoimmunization, antepartum haemorrhage, or a history of threatened miscarriage should be allowed to go significantly beyong term.

In an uncomplicated pregnancy no active intervention is called for if there is any doubt as to duration of the pregnancy, especially if the baby appears to be small. Otherwise, with a normal-sized baby and confidence in the period of amenorrhoea one ought to have good indications for *not* inducing labour.

In cases of cephalo-pelvic disproportion or with foetal distress, delivery by a Caesarean section will need to be considered.

H. The Elderly Primigravida

Definition:

The elderly primigravida is defined as a woman over the age of 35 years who is having her first pregnancy.

Significance:

The first pregnancy in a woman over 35 years of age is important because:–

a. Progressive conditions such as hypertension, rheumatic heart disease, and chronic bronchitis become worse with age.

b. Likelihood of future pregnancies is less.

c. Certain complications are more common, including:—

- Abortion
- Hyperemesis
- Fibromyomata
- Pre-eclampsia
- Accidental haemorrhage
- Premature labour
- Prolonged labour
- Higher perinatal mortality
- Higher maternal mortality

Management:

Antenatally more frequent visits are required with close attention to pre-eclampsia and evidence of poor foetal growth. At 36 weeks amenorrhoea assessment of pelvic dimensions is made. *Any* contraction of the pelvis is an indication for elective Caesarean section, since a trial of labour should have no place here. The finding of a malpresentation also warrants a Caesarean section. Prolonged pregnancy is avoided. At term there is no reason to avoid induction *if this is indicated.* During labour foetal distress is more common and monitoring will be required.

I. The Grand Multipara

Definition:

This term refers to a woman who has had five or more viable pregnancies.

Significance:

A special label is attached here to emphasise that these cases require special treatment due to increased risks to mother and baby. A number of complications is more common in this group of patients.

- Abortion is more frequent
- Anaemia is very common (Fe deficiency)
- Minor ailments, such as varicose veins or hiatus hernia are more common
- Multiple pregnancy is three times as common, reflecting these patients' high fertility
- Pre-eclampsia is slightly more common
- Accidental haemorrhage is doubled in incidence, and maternal mortality is increased seven-fold as a consequence of this.
- Rh isoimmunisation is more likely and severe
- Malpresentations are more common with the more lax abdominal walls, and predispose to a greater incidence of cord prolapse.
- Ruptured uterus is much more common, due to the:

i. fibrotic nature of the uterus.

ii. increase in malpresentations, and

iii. more forceful contraction, even in presence of obstruction.

- Pulmonary embolism is more common.
- Post-partum haemorrhage is more frequently met with.

Management:

1. Dietary supplements of iron, folic acid, Vitamin B complex, protein and calcium are needed.
2. Unstable or transverse lie at the 38th week must be treated by admission to hospital.
3. The possiblity of obstructed labour must never be forgotten.
4. Oxytocin must be used with the greatest caution (if at all) to induce labour, and *never* once labour has become established in a grand multipara.
5. Intravenous ergometrine is necessary for the third stage.
6. Contraception or tubal ligation ought to be offered, not merely supplied on request – the patient may never have heard of them.
7. The patient's anxiety lest she not get to hospital in time may lead her to be admitted in false labour. This will require tactful reassurance.

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CHAPTER 9

COMPLICATIONS OF THE PUERPERIUM

General Instructional Objective

Recognises and understands complications of the puerperium so that correct management can be instituted.

Specific Behaviours

1. Describes complications of the puerperium.
2. Demonstrates an ability to assess patients in the puerperium.
3. Discusses management of complications of the puerperium.
4. Demonstrates an ability to counsel a patient in the puerperium.
5. Discusses the aetiology of complications of the puerperium.

Complications of the Puerperium

SECTION A:
1. Post partum haemorrhage.
2. Inversion of the uterus.

SECTION B:
1. Puerperal infection.
2. Breast enlargement, cracked nipples and breast infection.
3. Urinary tract infection.
4. Vascular complications – thrombo-phlebitis and pulmonary embolism.

5. Haemorrhage.
6. Nervous system disorders.
7. Abnormalities of pelvic joints.

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SECTION A:

1. **Post Partum Haemorrhage** – is bleeding in excess of 300 mls from the genital tract during and after the third stage of labour. Some define it as a loss in excess of 600 mls, considering 300 mls to be comparatively normal.

Classification:

i. *Primary* – i.e. occurring within the first 24 hours of delivery.
   a. Atonic – from the placental site (80%).
   b. Traumatic – trauma to the genital tract (uterus, vagina, vulva, perineum) – 20%.
   c. Blood coagulation defect.

ii. *Secondary* (puerperal) – occurring between 24 hours and 6 weeks after delivery.

Primary Post Partum Haemorrhage

a. *Primary atonic post partum haemorrhage*

This is the commonest type of post partum haemorrhage and is due to lack of tone in the uterine muscle. Normally haemostasis is achieved by contraction and retraction of the muscle fibres, resulting in a reduction of size of the placental bed and "clamping" of the vessels in the uterine wall. If contraction of the uterus is prevented by retained placental frgaments or contraction and retraction fails to take place despite an empty uterine cavity, then haemorrhage will occur. An *empty, contracted* uterus will not bleed (from the placental site).

Factors predisposing to atonic post partum haemorrhages

i. *Those causing atony:*
   a. prolonged labour,
   b. prolonged analgesia and anaesthesia (especially halothane),
   c. multiparity,

   d. anaemia.

ii. *Those interfering with contraction and retraction:*
   a. Mismanagement of the third stage – by injudicious massaging and mishandling, distended bladder, more frequent retained fragments,
   b. retained placenta (or fragments),
   c. fibromyomata.

iii. *Other factors:*
   a. antepartum haemorrhage – placenta praevia, or concealed accidental haemorrhage,
   b. multiple pregnancy,
   c. previous post-partum haemorrhage.

Symptoms and Signs:

i. *Bleeding:*
   a. following onset of normal placental separation leading to a "latent interval" (minutes after delivery),
   b. comes in gushes, corresponding with uterine contractions.

ii. *Big, relaxed uterus:*
   a. soft uterus which is difficult to palpate,
   b. uterus rises above the umbilicus (even in absence of revealed bleeding).

iii. *Signs of Haemorrhage:*
   a. tachycardia, above 100/minute,
   b. hypotension,
   c. pallor,
   d. sweating,
   e. increased respiratory rate.

Management:

i. *Prophylactically*
   a. *in pregnancy* – correct pre-existing anaemia.
   b. *first stage of labour* –
      . careful observations,
      . correction of dehydration and ketosis,
      . adequate analgesia.

c. *second and third stages of labour* –
   . oxytocin 5 units IMI with delivery of anterior shoulder,
   . ergometrine 0.5 mg IMI immediately following delivery of placenta (in absence of hypertension above 140/90 mm Hg.)

Note: i. *If assisted delivery* – oxytocin is given intravenously with delivery of anterior shoulder.

ii. *If previous post partum haemorrhage or pre-disposing complication* – erogmetrine 0.25 mg IVI is given with the delivery of the anterior shoulder. After the placenta is delivered 50 units of oxytocin should be added (and well mixed) to the bottle of IV fluids set prior to the second stage of labour as a precautionary measure.

iii. *For patients significantly at risk* – in addition to ii. above, blood should be cross-matched.

Treatment of Post-partum Haemorrhage:

a. *Contract the uterine muscle by:*

   . ergometrine 0.5 mg IVI (may repeat in 1 hour),
   . fundal massage,
   . express clot/placenta in lower segment,
   . bimanual compression of uterus (if necessary).

b. *Treat shock:*

   . raise foot of the bed,
   . ensure airway is clear,
   . remove blankets (to stimulate vasoconstriction),
   . cross match blood (at least 2 bottles),
   . commence intravenous therapy (preferably with S.P.P.S.),
   . give blood as soon as it is available.

c. *Ensure uterus is empty:*

   . check that delivered placenta is complete,
   . if retained, deliver by Brandt-Andrews manouvre (if uterus is contracted) or manual removal.

d. A nurse must remain with the mother, observing closely her colour, pulse, blood pressure, vaginal loss and fundal height over the next 2-3 hours. If these observations prove normal and the maternal condition is satisfactory, the patient

may then be transferred to a ward. Close observation must be maintained for another 12 hours at least.

Summary:

1. If the bleeding can be controlled quickly by ergometrine and massage, this may be all that is necessary.
2. If the uterus is not empty but the bleeding is quickly controlled, then treat shock (if present) and arrange manual removal when the patient is resuscitated.
3. If the uterus is not empty and bleeding is not controlled, then manual removal must be performed immediately with resuscitation beginning at the same time. Unless an anaesthetist is ready and waiting, N_20 and oxygen is sufficient for this emergency procedure.

b. *Primary Traumatic Post-Partum Haemorrhage*

Aetiology:

Trauma following –
i. rapid delivery,
ii. episiotomy,
iii. instrumental delivery.

Symptoms and Signs:

i. *Bleeding:*

   a. occurring immediately (i.e. no latent period),
   b. continuous or increasing with maternal respiration.

ii. *General signs of haemorrhage* – shock, etc.

iii. *Uterus – firm, contracted.*

iv. *Haematoma formation* in abdomen or genital tract.

Management:

i. *Prophylaxis* – good obstetrics.
ii. *Treatment* –

a. *Stop the bleeding* (temporary measures):

   . pressure with a sterile pad,
   . tight vaginal pack (rarely needed).

b. *Suturing:*

. determine extent of trauma (including examination of cervix),
. lithotomy position,
. adequate light, analgesia,
. competent assistant.

c. *Resuscitation:*

. when needed, either before or during suturing.

d. If *genital tract haematoma* is present (may be manifest several hours after delivery):

. general anaesthetic (usually),
. evacuation of haematoma,
. suture of bleeding vessels,

c. *Blood Coagulation Defect*

If the uterus is empty and contracted and there is no trauma to the genital tract, excessive bleeding may not occur even in the presence of a coagulation defect.

Secondary Post Partum Haemorrhage

Incidence:

Approximately 1/900, occurring most frequently between the 5th and 15th days after delivery.

Aetiology:

i. Infection of retained placental fragments.
ii. Infection of fibroids (especially fibroid polyps).
iii. Infection of a wound in the genital tract.
iv. Chorionepithelioma.
v. Fresh trauma.

Mamagenent:

i. *Prophyaxis:*

a. Ensure uterus is empty following delivery.
b. Check that delivered placenta is complete.
c. Early and adequate treatment of any infection.

ii. *Treatment:*

a. If less than 48 hours after delivery – fundal massage.
b. Ergometrine 0.5 mg – may be repeated.
c. Resuscitation if necessary.
d. Antibiotics.
e. Removal of placental fragments – digital exploration or light curettage.
f. Suturing or resuturing of wound.

Retained Placenta and Placental Fragments

Definition – placental tissue retained in the uterus for more than 30 minutes following the birth of the baby.

Aetiology:

i. *Mismanagement* of the third stage –

a. Meddlesome manipulation of the fundus.
b. Oxytocics given too early.

ii. *Constriction* ring forming at the junction of the upper and lower uterine segments may prevent the descent of a separated placenta. It may occur spontaneoulsy or follow injudicious handling of the fundus.

iii. *Adherent placenta* –

a. *Simple* adherent placenta.
b. *Placenta accreta* (rare). The chorionic villi penetrate the muscle layer resulting in pathological adherence of the placenta to the uterus. The condition may either bleed profusely (partial placenta accreta) or not bleed at all (complete placenta accreta).

Management:

i. *Prophylaxis* – good obstetrics, especially with patients who have a history of previous post-partum haemorrhage or retained placenta.

ii. *Treatment:*

a. Inspect placenta soon after delivery. Note if it is complete or if there is a succenturiate lobe retained.

b. Manual removal of placenta or fragments.

c. If *bleeding present* – treatment as outlined for post partum haemorrhage.

Note: With partial placenta accreta haemorrhage may be uncontrollable necessitating hysterectomy.

d. If *bleeding absent* –

i. *Simple adherent placenta* –
. attempt Brandt-Andrews manouvre (if uterus contracted),
. manual removal with adqeuate anaesthesia followed by ergometrine 0.5 mg IVI (may repeat in 4 hours).

ii. *Complete placenta accreta* – placenta is left in utero.

e. Careful observation (in the "lying-in" wards) of those patients with an abnormal third stage.

2. **Acute Inversion of the Uterus**

Definition:

The uterus is turned inside out (either partially or completely).

Incidence:

Rare (1 in 10,000).

Aetiology:

i. *Spontaneous* – with fundal attachment of placenta.

ii. Excessive fundal pressure.

iii. Excessive cord traction before the placenta has separated or when the uterus is incompletely contracted.

Sympyoms and Signs:

i. Shock,

ii. Fundus not palpable abdominally,

iii. Vulval tumour,

iv. Pain (may be severe),

v. Bleeding (unless placenta is still adherent).

Management:

i. *Prophylaxis* – good obstetrics.

ii. *Treatment:*

a. Resuscitate (if necessary).
b. Immediately *replace* uterus (may need anaesthetic) by –
. gentle manual pressure,
. hydrostatic, i.e. uterus is replaced into the vagina, which is then filled with warm saline (with reservoir at 2 feet), *or* Huntington's operation (laparotomy).
c. Oxytocics (intravenously) – to produce uterine contraction and to prevent a recurrence.

SECTION B:

Complications of the Puerperium

Before considering the complications of the peurperium it would be beneficial to revise the course and assessment of the normal puerperium (Chapter 2).

1. *Puerperal infection* is pyrexia during the puerperium due to an infection of the genital tract or its adnexae. Prior to the advent of antibiotic therapy it was one of the most common causes of maternal death.

During the puerperium the female genital tract is particularly susceptible to infection as –

i. There is a raw placental site (equivalent to a superficial wound.).

ii. Wounds may occur in the lower birth canal (cervix, vagina, fourchette).

iii. The lochia are alkaline and increase vaginal pH.

iv. Bruised and devitalised tissue or blood clots may provide a favourable environment for infection.

v. The stresses of pregnancy lower resistance – anaemia, long labours with added interference, socio-economic factors (diet and nutrition).

The organisms responsible for infections –

a. In the upper genital tract –
. Coliform organism,
. Streptococcus faecalis,
. Anaerobic streptococcus,

. Haemolytic streptococci, Groups A and B.
. Clostridium welchii.

b. In the lower genital tract –
. Coliform organisms,
. Staphylococcus aureus.

Infection may be *endogenous* (caused by organisms present in the genital tract prior to labour), *autogenous* (from elsewhere in the patient's body), or *exogenous*. The majority of cases are caused by potential pathogens which normally inhabit the vagina (especially anaerobic streptoccoci) or the intestine (E. coli.)

Diagnosis:

i. *History* of predisposing factors –

. antenatally – anaemia,
diabetes.
. during confinement –
protracted labour,
operative interference,
episiotomy,
retained fragments.

ii. *Symptoms and Signs:*

. fever ± rigors,
. high fundus (congested),
. lower abdominal discomfort and/or pain,
. foul purulent lochia,
. bimanual examination to detect tenderness or swelling of the uterus or tubes.

Management:

i. *Prophylaxis:*

Asepsis and antiseptics.

ii. *Notification:*

Any febrile condition occurring in the first ten days of the puerperium in which a temperature of 38°C or higher appears upon more than one day during that period, is considered to be puerperal infection and is notifiable.

iii. *Investigation:*

. High vaginal swab (culture and sensitivity),
. FBC (especially Hb and WCC),
. MSU,
. Chest X-ray,
. Blood cultures.

iv. *Treatment:*

. Local heat for infection of vulva and vagina,
. Removal of sutures,
. Antibiotics – penicillin 1 mega unit 6 hourly,
streptomycin 0.5 gm 8 hourly, until bacterial sensitivities are available.
These should be changed if the organisms determined are not sensitive *and* the patient is not clinically improving.

Puerperal infection may include that *localised* to the vulva and vagina, cervix or endometrium and myometrium or *spreading* infection (from the uterine cavity or cervix) causing salpingitis, pelvic cellutis, peritonitis or septicaemia.

Specific infections: a. Clostridium welchii,
b. Gram-negative septicaemia.

a. *Clostridium welchii infection:*

This organism causes "gas gangrene". It may also be present in the vagina without being pathogenic. It is anaerobic and grows in dead tissue.

Diagnosis:

. pain,
. high temperature,
. shock,
. haemolytic anaemia (with jaundice),
. "port-wine" urine – haemolysis,
. anaerobic blood culture.

Treatment:

i. Penicillin 1 mega-unit four-hourly, and polyvalent antiserum.

ii. Electrolytes (acidosis may occur and must be corrected if present),
blood transfusion may be necessary,

haemodialysis may be required.

iii. Hydrocortisone 2 gms IV – may be repeated in 2 hours if necessary.
Other measures to treat shock (due to peripheral circulatory failure – endotoxic shock).

iv. Surgery – empty the uterus when the patient is resuscitated and the blood pressure stabilised.

v. Central venous pressure manometer.

b. **Gram-negative septicaemia:**

Diagnosis:

. pallor with cold extremities,
. anxious and sweating,
. abdominal pain and rigid abdomen,
. a petechial rash may be noticed prior to onset of symptoms,
. blood and urine culture,
. vaginal and cervical swabs.

Treatment:

i. Ampicillin 500 mgs 4-hourly,
ii. Detection and treatment of acidosis (if severe this may lead to cardiac arrest),
iii. Prophylactic digitalisation may be instituted,
iv. Hydrocortisone,
v. I.V. fluid to maintain circulating blood volume,
vi. Central venous pressure manometer.

2. Breast Engorgement, Cracked Nipples and Breast Infection

i. *Breast engorgement* is an important condition to recognise as it may predispose to mastitis and breast abscess, as well as being a very painful condition in itself.

Aetiology:

In establishing a balance between maternal milk production and demand by the infant milk secretion may easily exceed removal. The breasts therefore become tense and over-distended and may obstruct venous and lymphatic drainage, thus aggravating the problem.

Clinically:

Breasts are full, hard and painful,
The patients may have a slight temperature.

Management:

a. Analgesics.
b. Adequate breast support.
c. Expression of milk – manual, breast pump ± syntocinon spray.
d. Suppression of lactation may be achieved with stilboestrol (15 mgs tds) – (becoming a less popular mode of treatment, and only if other measures fail).

ii. *Cracked Nipples:*

Aetiology:

Aggressive sucking by baby and a tendency to bite rather than suck especially if nipple is inverted.

Importance:

May allow entry of bacteria.

Management:

a. Rest the nipple,
b. Use of nipple shield,
c. Local administration of antiseptics – hexachlorophene.

iii. *Puerperal mastitis and breast abscess:*

Aetiology:

Bacteria (most often Staphylococcus aureus) entering the interstitial tissues via cracked nipples.

Clinical:

. Fever ± rigors,
. Painful breast,
. Reddened tender segment of breast which may become *acutely* tender, occurring 1-3 weeks after delivery.

Management:

i. *Prophylaxis:*

. treat breast engorgement,
. care with feeding and cracked nipples.

ii. *Treatment:*

. antibiotics (note – organisms are usually penicillin resistant),
. surgical drainage of abscess for 3 days (pus is sent for culture and sensitivity) – do not wait for sign of fluctuation,
. infra-red heat to aid healing.

3. **Urinary Tract Infection**

This is the second most common infection of the puerperium. The majority are caused by E. coli.

Predisposing causes:–

i. Infection often occurs in pregnancy and then recurs in the puerperium. Urinary stasis is encountered as an effect of *progesterone* on the bladder and ureters – large amounts of urine may be held without feeling a need to empty the bladder.

ii. *Catheterisation* during and after labour may introduce bacteria

Diagnosis:

i. *Clinical Features:*

. fever ± rigors,
. backache or loin pain,
. lower abdominal pain,
. dysuria and frequency,
. nausea and vomiting.

ii. *Investigations:*

. vaginal swab and culture,
. MSU – microscopy and culture,
. Hb and WCC.

Treatment:

Appropriate antibiotics, e.g. Bactrim 1 tablet bd., or Gantrisin 2G 6 hrly.

4. **Vascular complications:**

Between 1-3% of women (in Australia) will develop some form of venous complications –

a. *Thrombophlebitis* – inflammation and thrombosis, usually of a superficial vein.

b. *Phlebothrombosis* – thrombosis usually of the soleal sinuses, the femoral vein or deep pelvic veins (ovarian, uterine and hypogastric veins). The danger lies in that, without an inflammatory component, the thrombus is poorly adherent to the vessel wall.

c. *Pulmonary embolism.*

Aetiology:

i. Stasis – bed rest,
varicose veins.

ii. Altered coagulability of blood (altered coagulation factors).

iii. Oestrogen given in puerperium to suppress lactation.

iv. Increase after Caesarean section or if previous thrombosis has occurred.

Diagnosis:

1. **Clinical Features:**

a. *Thrombosis*

. often none (until pulmonary embolism occurs),
. slight temperature,
. may get swollen calf (measure and compare with other leg and with any previous measurements),
. swollen ankle (unilateral),
. tender, painful calf,
. positive Homan's sign – an unreliable indication of the presence of deep thrombosis.

b. *Pulmonary Embolism*

. may be asymptomatic (small emboli are thought to be as common as deep venous thrombosis),
. if large – sudden collapse,
tachycardia,
dyspnoea,
pleuritic chest pain) if pulmonary
haemoptysis) infarction
pleural friction rub) occurs.

Differential diagnosis – cardiac infarction, septic shock.

2. **Investigations:**

a. *Thrombosis*

. Ultrasonic detection,
. Venography occasionally, or
. Radiofibringogen – if used early; it must be incorporated in the head of the thrombus.

b. *Embolism*

. chest X-ray,
. ECG,
. lung scan,) in special surgical units.
. pulmonary angiography)
in addition to investigations for thrombosis.

Management:

a. *Thrombosis*

i. elevate foot of bed to increase venous return, local Hirudoid or Lasonil ointment to relieve discomfort, mild analgesics,
watch for extension.

ii. intravenous heparin monitored by clotting times.

b. *Embolism*

i. *heparinisation* – 10,000 units stat
5,000 units 4-horuly
by intravenous infusion for 7 days.

ii. *streptokinase infusion* – this is a dangerous substance, so that its use is severely restricted. It lyses all clots in wounds and is absolutely contraindicated within 5-6 days of surgery. It may be infused by catheter into the pulmonary artery.
Fibrinogen levels and fibrin degradation product levels must be monitored to determine the coagulation status of the patient.

iii. *Surgery*

. cardiopulmonary bypass,
. embolectomy,
. ligation of veins or plication of the inferior vena cava may be necessary if emboli are recurrent.

Prophylaxis:

Early ambulation, especially after Caesarean section.
Treat early indications and signs of thrombosis.

5. **Haemorrhage** – discussed in SECTION A.

6. **Psychiatric considerations**

The popular conception of complete contentment for the mother and expansive pride on the part of the father, in the immediate post partum period, is all too often incorrect. Too many women lack confidence to handle their infant with complacence and the problems associated with breast feeding may lead to tremendous feelings of depression and inadequacy if handled unsympathetically. Insomnia is almost universal and night time sedation is most important. This may be achieved by prescribing 5-10 mg Mogadon nocte. In general, mental illness is considered as being latent and only precipitated by pregnancy.

Management:

It is preferable for a woman with a history of mental illness to be seen by a psychiatrist during the ante-natal period.

A phase of acute depression between the third and fifth day is so constant that it might be better to warn the patient of the possiblity. When it occurs, a quiet talk for 10-15 minutes is usually necessary to reassure the patient, particularly stressing that it is a normal reaction and a transitory phenomenon. Faulty handling at this stage can produce a state of chronic anxiety, characterised by fatigue, depression, extreme lack of confidence and rejection of baby and husband.

In about 1 in a 1,000 puerperal women this will progress to a true puerperal insanity, half of which cases will occur within the first fortnight, To the above symptoms will be added irritability, headache and lability of mood, quickly giving way to suspiscion, confusion, incoherence, nightmares, anxiety over minutia and refusal of food. Many cases will develop a delerium and excitement phase and ultimately delusions and complete mania. Suicidal and infanticidal urges are not uncommon.

If any abnormal behaviour is noticed early in the puerperuim the patient should be nursed in a single room and conservative management with sedatives and tranquilisers is at first instituted.

If no improvment occurs within a week, transfer to a psychiatric unit should be considered. Psychiatric treatment is usually successful but there is a high relapse rate after initial improvement and many cases remain permanently deranged.

7. *Abnormal separation* of the symphsis pubis or sacroiliac joints usually settles in 7-10 days.

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CHAPTER 10

ABNORMAL PRESENTATION AND MULTIPLE PREGNANCY

General Instructional Objective
Recognises abnormal presentations or multiple pregnancies and understands their significance so that he can manage the patient appropriately.

Specific Behaviours

1. Demonstrates an ability to recognise abnormal presentations or multiple pregnancies.
2. Discusses aetiological factors causing abnormal presentations or multiple pregnancies.
3. Describes the complications which may occur with abnormal presentations and discusses their management.
4. Describes the complications which may occur with a multiple pregnancy and discusses their management.
5. Discusses the management of women with multiple pregnancy and abnormal presentation.
6. Demonstrates technique of breech delivery on a mannikin.

Abnormal Presentation and Multiple Pregnancy

The following conditions will be described:

. Face presentation.

. Brow presentation.
. Median vertex presentation – The Military attitude.
. Breech presentation.
. Transverse lie (shoulder presentation).
. Compound presentation.
. Mulptile pregnancy.
. Prolapsed cord.

A. Face Presentation

Definition:

Presentation	–	cephalic
Presenting part	–	face
Attitude	–	complete extension
Denominator	–	mentum
Pres. diameter	–	submento-bregmatic (9.5 cm).

Incidence:

1/500 births. Most are secondary, i.e. taking place during labour. About 70% are anterior or transverse, e.g. L.M.A. (left mentum anterior), and 30% are posterior.

Aetiology:

Some 70% occur by chance, as a change in attitude from incomplete flexion to extension at engagement. Rarer causes include thyroid neoplasms, multiple coils of cord around the neck, monsters, anencephalic foetuses, and conversions from brow presentation.

Diagnosis:

Diagnosis is rarely made before labour. Because of good progress most cases are diagnosed late in established labour. On abdominal palpation in a L.M.T. position, for example, the occiput may be very prominent with a deep groove between it and the smooth back. On vaginal examination a clue is the *absence* of a round, even, hard vertex. There is a soft and irregular part to be felt. One suspects a face, a breech, a compound presentation, or a monstrosity (after prolonged labour oedema may confuse the picture). Gentle palpation of the orbital ridges and eyes, irregular nose, and mouth, will confirm the diagnosis. An X-ray examination is performed to both confirm the diagnosis and estimate pelvic capacity.

Management:

The majority of face presentation engage at the brim in the transverse diameter of the pelvis, R.M.T. more commonly than L.M.T. (Fig. 10.1) Because of the small presenting diameter (9.5 cm) most come to spontaneous delivery or delivery by outlet forceps (Fig. 10.1). About 30% of face presentations are posterior. Most of these rotate anteriorly to the mento-anterior position (Fig. 10.1-D) and proceed spontaneously. Persistent posterior face presentations cannot deliver spontaneously and operative procedures are necessary. The following may be attempted:

1. *Flexion* to an occipito anterior position. This usually fails and is not advised.
2. *Rotation* to a mento-anterior position from a mento-posterior position (manually or with forceps), e.g. Kiellands (is difficult and rarely successful).
3. *Lower segment Caesarean section* (L.S.C.S.) is probably the best management for the established posterior face presentation.
4. *Destructive Operation* when the baby is dead (see page 4.13).

Complications:

Maternal morbidity is increased with any operative interference (20%).
Foetal risks are cerebral congestion, hypoxia due to poor venous return, and increased morbidity due to operative procedures.

B. Brow Presentation

Definition:

Presentation	–	cephalic
Presentaing part	–	sinciput (brow)
Denominator	–	frontum, e.g. L. Fr. A. (left frontum anterior).
Presenting diam.	–	mento-vertical (13.5 cm) (Fig. 10.2).

Incidence:

Less than 1/1000 births. Often the position is transitory flexing to an occiput posterior position, or extending to a face.

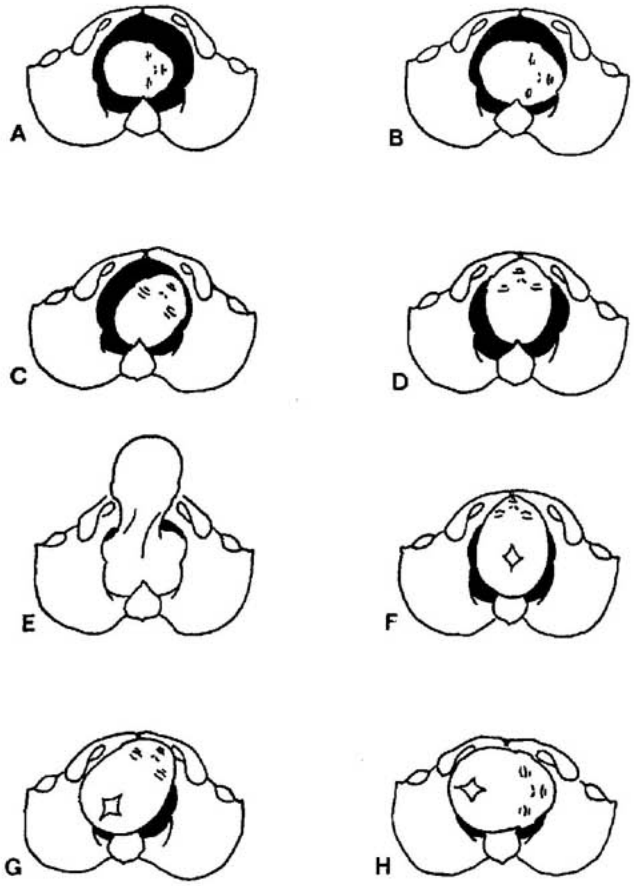

Fig. 10.1. Mechanisms of labour in face presentation. The head engaged the brim in the left, mento-transverse position (L.M.T.).
A. L.M.T. onset of labour.
B. Descent.
C. Internal rotation: L.M.T. to L.M.A.
D. Internal rotation: L.M.A. to M.A.
E. Birth by flexion.
F. Extension.
G. Restitution: M.A. to L.M.A.
H. External rotation: L.M.A. to L.M.T.

Aetiology:

As for face presentation.

Diagnosis:

It is difficult to diagnose on abdominal palpation and is rarely diagnosed prior to the establishment of labour. Abdominally the head is not engaged (unless the pelvis is large and the baby is small) and the occipital prominence is at the back. Vaginally palpation of the supraorbital ridges and the anterior fontanelle is the key to diagnosis. Diagnosis is difficult and radiology may be needed.

Fig. 10.2. Brow presentation. The mento-vertical diameter (13.5 cm) cannot engage the pelvic inlet.

Management:

Only 10% will either convert spontaneously or be converted to a *face* or *vertex* presentation and deliver spontaneously. A short trial of labour is permissible therefore, with early attempt at flexion or extension. 90% will *not* deliver spontaneously and Caesarean section must be carried out.

Complications:

A neglected brow presentation may result in prolonged and

traumatic labour. Perineal tears may be extensive or rupture of the uterus may occur and foetal mortality is high from excessive moulding.

C. Occipito-Posterior Median Vertex Presentation:

"The Military Attitude"

(This is a common cause of delay in the 2nd stage of labour)

Fig. 10.3. Median Vertex Presentation: Military attitude. The occipito-frontal diameter engages the pelvic brim (11.5 cm).

Definition:

Presention	–	cephalic
Presenting part	–	vertex (median vertex)
Denominator	–	occiput (usually posterior)
Diameter	–	occipito-frontal (11.5 cm), (Fig. 10.3).

Diagnosis:

Vaginally both fontanelles are at the same level in the pelvis.

Management:

In some cases the military position is transitory and as the head descends it flexes. The labour is a little longer and harder but the prognosis is good. Where the head extends to brow or face presentation the management is as of the resulting presention. Arrest with persistent military attitude should be treated by flexing the head, rotationg the occiput anteriorly, and extracting by forceps (see Deep Transverse Arrest).

D. Breech Presentation

Definition:

Lie	–	longitudinal
Presentation	–	breech (buttocks lie over pelvis brim)
Presenting part	–	buttocks, feet, or knees
Denominator	–	sacrum, e.g. Left Sacrum Anterior (L.S.A.)

Incidence:

About 3% of all deliveries.

Aetiology:

In the normal pregnancy before the 32 nd week of gestation the foetal position is unstable because of a relative excess of liquor. The position may therefore readily interchange between cephalic and breech presentation.

- Foetal movements favour instability.
- Foetal kicking promotes the movements of the breech away from the firm maternal bony pelvis into a more yielding uterine fundus so that foetuses with extended legs, unable to kick against the maternal brim, often remain as a breech presentation.
- The larger breech will tend to remain in the larger fundus, the head in the smaller pelvic end.

A persistent breech is therefore more likely with:

1. Prematurity – smaller baby and more liquor.
2. Factors interfering with spontaneous version –
   a. twins,
   b. oligohydramnios,
   c. extended legs,
   d. uterine abnormality,

e. fibromyomata.

3. Unknown reasons (22%).

Associated Conditions my be –

a. Hydramnios – free, movement of foetus

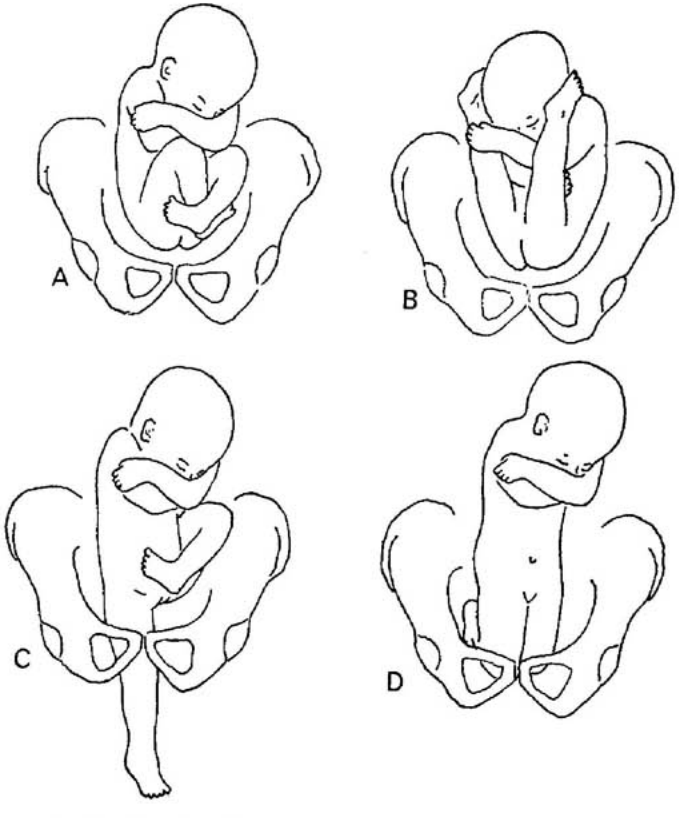

Fig. 10.4. Classification of breech presentations.
A. Complete breech.
B. Frank breech.
C. Footling breech (single).
D. Kneeling breech (double).

b. Lax abdominal wall,

c. Placenta praevia because –

i. the pelvic mass keeps the presenting part high and to one side,
ii. premature labour is more common.

Classification (Fig. 10.4):

1. *Full* (complete) *Breech* –

   Legs are flexed at the thighs and knees.

2. *Frank Breech* –

   Legs are flexed at the thighs and extended at the knees. The most common form (more than 60% of all breeches).

3. *Footling Breech* –

   May be single or double. There is extension at thighs and knees. The foot is the leading part.

4. *Kneeling Breech* –

   May be single or double. There is extension at the thighs and flexion at the knees.

Management of Breech Presentation During Pregnancy:

Since mortality due to breech presentation is at least double of vertex presentation, it is important that the presentation be detected *before week 34* so that external version may be attempted.

Diagnosis:

1. The patient may complain of discomfort under the ribs and may feel a lump in the epigastrium.
2. On palpation –
   . the *lie* is longitudinal,
   . a round, hard, regular mass (head) is in the fundus,
   . in the pelvis a soft, irregular mass is found.
3. Foetal heart sounds may be above the umbilicus. This not a rule.
4. If in doubt a vaginal examination will reveal an irregular and soft mass with no suture lines lying in the pelvic inlet. Detect the anus and ischial tuberosities.
5. If still in doubt an X-ray should be taken.
6. Look for the *cause* of the breech (see *Aetiology* p. 10.4).

External Ceplalic Version:

This is a manoeuvre by which the foetal polarity is changed to a cephalic presentation, all manipulations being done through the abdominal wall. It will reduce the incidence of mature breech delivery by 1%. It should be performed between the 32nd and 34th weeks. If carried out before 32 weeks, many babies revert to the breech. If carried out after 34 weeks the manipulation becomes more difficult because of relative reduction in the liquor amnii.

Contra-indications to Version include:

1. Maternal disease – hypertension or pre-eclampsia; diabetes mellitus; rhesus incompatibility.
2. Antepartum haemorrhage.
3. Twins.
4. Previous classical Caesarean section (Uterine corpus incised).
5. Where Caesarean section is undertaken for other reasons.
6. Ruptured membranes.
7. Intrauterine death.

Technique of External Cephalic Version

Technique of External Cephalic Version

Disengage the breech from the pelvis. Exert gentle pressure on each pole of the foetus maintaining flexion, until the poles are past the transverse position. Check foetal heart sounds throughout the version, and if any irregularity occurs reconvert the lie to the original. If the breech recurs never carry out version more than twice.

Dangers of External Cephalic Version

1. Foetal distress due to cord entanglement.
2. Separation of the placenta leading to antepartum haemorrhage and intrauterine death.
3. Intrauterine death (often without explanation).

4. Premature rupture of the membranes with onset of premature labour.
5. Possibility of release of foetal Rh + ve cells into the maternal Rh — ve circulation.

Radiology:

If external cephalic version is not attempted or fails, X-ray pelvimetry must be carried out to determine whether the after-coming head is likely to pass through the pelvic brim.

Delivery

Three methods of approach are available.

1. To allow labour to start spontaneously.
2. To induce labour before or at the E.D.D. and deliver a smaller baby.
3. To perform a Caesarean section.

Most patients should come into labour spontaneously. Induction may result in prematurity or cord prolapse. Caesarean section is reserved for complicated 'breeches' with –

a. suspected disproportion,
b. antepartum haemorrhage,

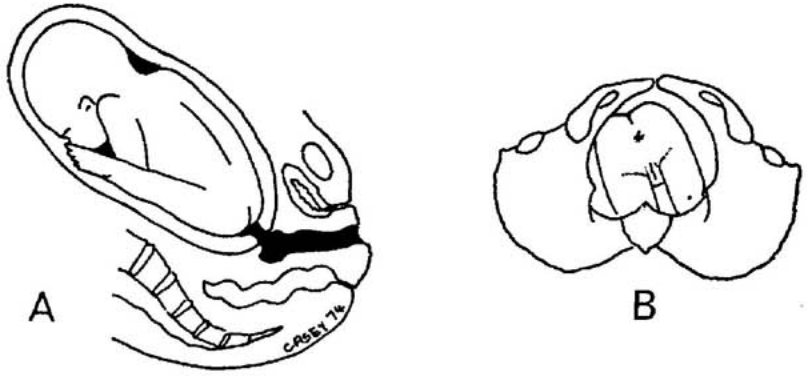

Fig. 10.5. The right sacral anterior position (R.S.A.). Onset of labour.

c. primigravida over 35 years of age,
d. severe hypertension,
e. delay in first stage,
f. cord prolapse.

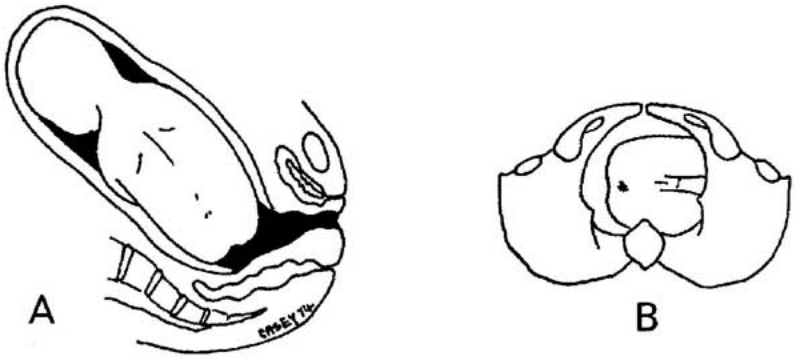

Fig. 10.6. The right sacral anterior position. Descent, lateral flexion and internal rotation of the buttocks.

Mechanisms of Labour

These may be subdivided into three sections. The Right Sacral Anterior position will be considered for an example (Fig. 10.5).

1. **Buttocks and Lower Limbs**

Descent:

Engagement takes place when the bitrochanteric diameter has passed the inlet. In the R.S.A. position the sacrum is in the right anterior quadrant of the pelvis. The bitrochanteric diameter is in the right oblique diameter. Descent is slow since the breech is a poor dilator of the cervix.

Flexion:

Lateral flexion at the waist following the pelvic axis.

Internal Rotation:

The leading anterior hip meets the resistence of the pelvic floor and rotates anteriorly towards the midline. The bitrochanteric diameter is now in the A.P. diameter of the outlet (Fig. 10.6.B).

Birth of Bottocks by Lateral Flexion:

The anterior buttock passes under the pubis. Lateral flexion occurs and the posterior buttock is born over the perineum. The feet should now be brought down (Fig. 10.7).

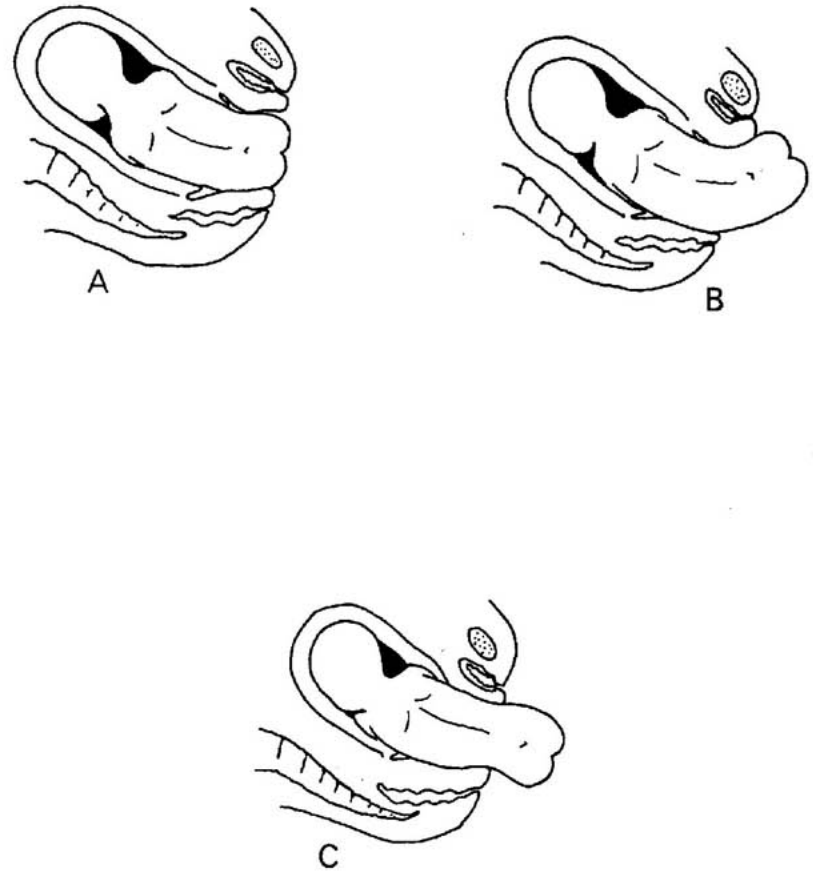

Fig. 10.7. Birth of buttocks by lateral flexion during breech delivery. Crowning of the breech occurs when the bitrochanteric diameter passes through the dilated perineum.
A. Breech crowning.
B. Birth of posterior buttock.
C. Birth of anterior buttock.

2. The Shoulders and Arms

Engagement:

Engagement of the shoulders takes place in the right oblique diameter of the pelvis (Fig. 10.8.A).

Internal Rotation of Shoulders:

The anterior shoulder rotates under the symphysis to place

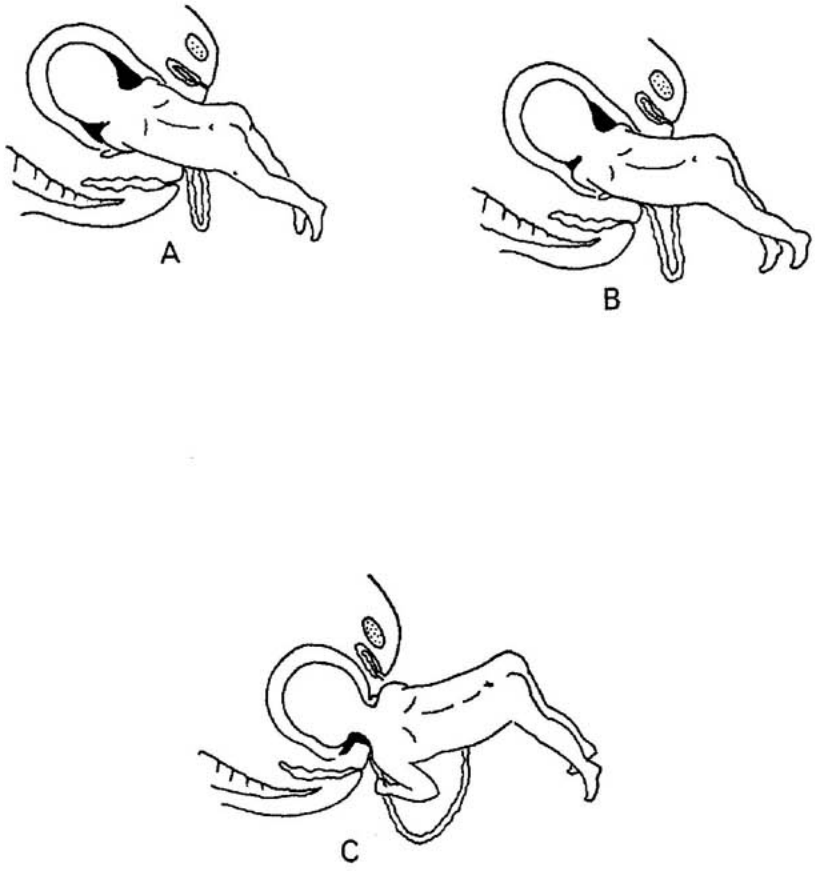

Fig. 10.8. Mechanism of shoulder and arm delivery in the breech presentation.
A. Feet born, shoulders engaging.
B. Descent and internal rotation of shoulders.
C. Posterior shoulder born; head has entered the pelvis.

the bisacromial diameter in the A.P. diameter of the outlet (Fig. 10.8.B).

Birth of Shoulders by Lateral Flexion:

The anterior shoulder impinges under the symphysis and the posterior shoulder and arm are born over the perineum as the baby's body is lifted, (Fig. 10.8.C). Lowering the baby allows the anterior shoulder to pass under the symphysis (Fig. 10.9.A).

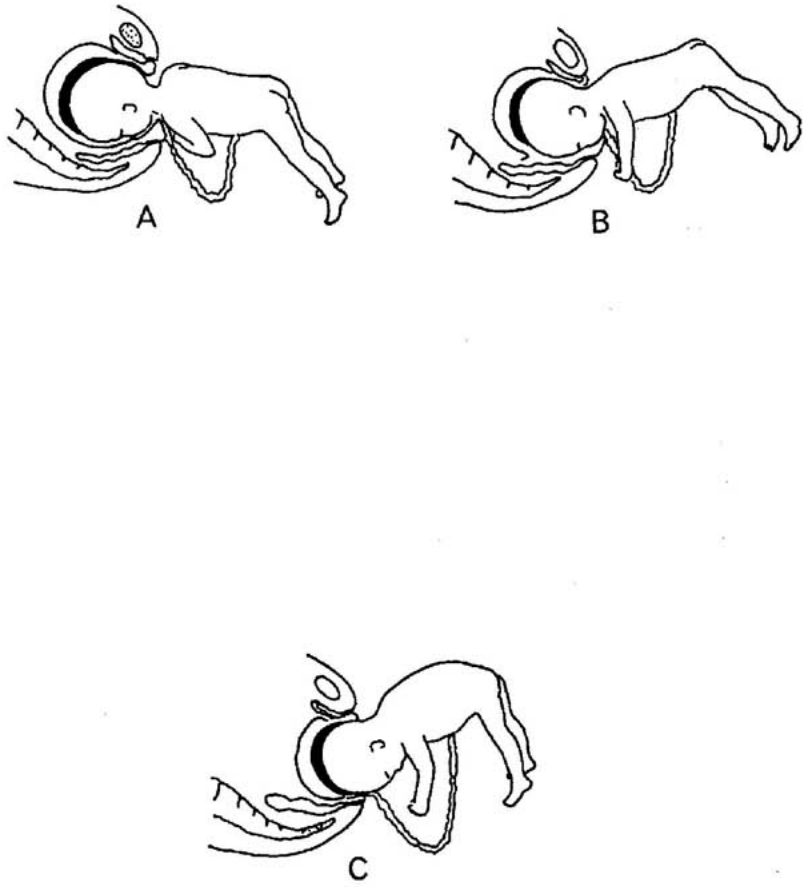

Fig. 10.9. Mechanism of the delivery of the head in a breech presentation.
A. Anterior shoulder born; descent of head.
B. Internal rotation and beginning flexion of the head.
C. Flexion of the head complete.

3. **The Head**

Descent and Engagement:

When the shoulders are at the outlet the head is entering the pelvis with the saggital suture in the left oblique diameter (Fig. 10.9.A).

Flexion:

Flexion now takes place as the uterus contracts on the head (Fig. 10.9 B. and C.)

Internal Rotation:

At the pelvic floor the head rotates to the A.P. diameter of the outlet, the occiput coming under the symphysis (Fig. 10.9.B).

Birth of the Head by Flexion:

The nape of the neck pivots under the symphysis and the face, brow, and the occiput are born over the perineum by flexion (Fig. 10.9.C).

Management of the Breech Delivery:

The mechanisms described above include no obstetrical assistance. This, in fact, rarely happens. Normally the baby is delivered as an "assisted breech" where the infant progresses by the natural forces as far as the umbilicus, and the remainder is extracted by the attendant. Total Breech Extraction, where the entire infant is extracted by the attendant, is indicated for the delivery of a second twin (see page 216), and less commonly for the management of prolapsed cord, and foetal distress after internal version.

1. **Management of First Stage of Labour** (Breech Presentation) Observations are the same as for a cephalic presentation except that more frequent observations of the foetal heart sounds are made (1 hourly), and a vaginal examination is performed to exlcude cord prolapse at the rupture of the membranes.

2. **Management of Second Stage of Labour** (Breech Presentation) The mother may now have a desire to "bear down" before full dilatation of the cervix has been reached. This should be delayed until delivery is possible.

   If the second stage is prolonged over 1 hour, the case must be reassessed with view to Caesarean section.

With normal progress at full dilatation the steps are (anaesthetist present and ready for general anaesthetic):

a. Patient is placed in the *lithotomy* position and prepared for delivery.

b. *Perineal infiltration* with 1/2% Xylocaine (less than 40 mls) in preparation for episiotomy.

c. The *bladder is catheterized.*

d. An *Episiotomy* is performed when the breech is distending the perineum, usually the cut is made on the opposite side to the infant's genitals.

e. *Nothing is done* to help the breech across the perineum until the umbilicus is seen. Pulling on the breech can extend the head and arms. The mother should be encouraged to bear down hard. Fundal pressure is of assistance.

f. As the umbilicus is born *pull down a loop of cord* (to see that it is long enough to avoid avulsing the cord from the abdomen. Now a maximum of 5 minutes are available before breathing must begin (to obviate brain damage) since the cord is being compressed (Fig. 10.10).

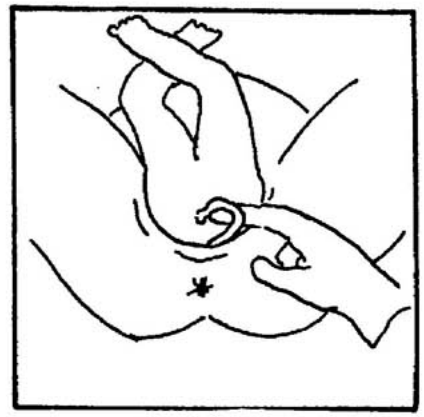

Fig. 10.10. Loop of umbilical cord being pulled down.

g. The *patient is anaesthetized.*

h. A warm towel is placed about the buttocks (not round the

abdomen since liver trauma is possible), and the *Lovset Manoeuvre* is performed for the delivery of the arms (Fig. 10.11). Keeping the baby's back to the mother's front at all times, a combination of rotation and down-

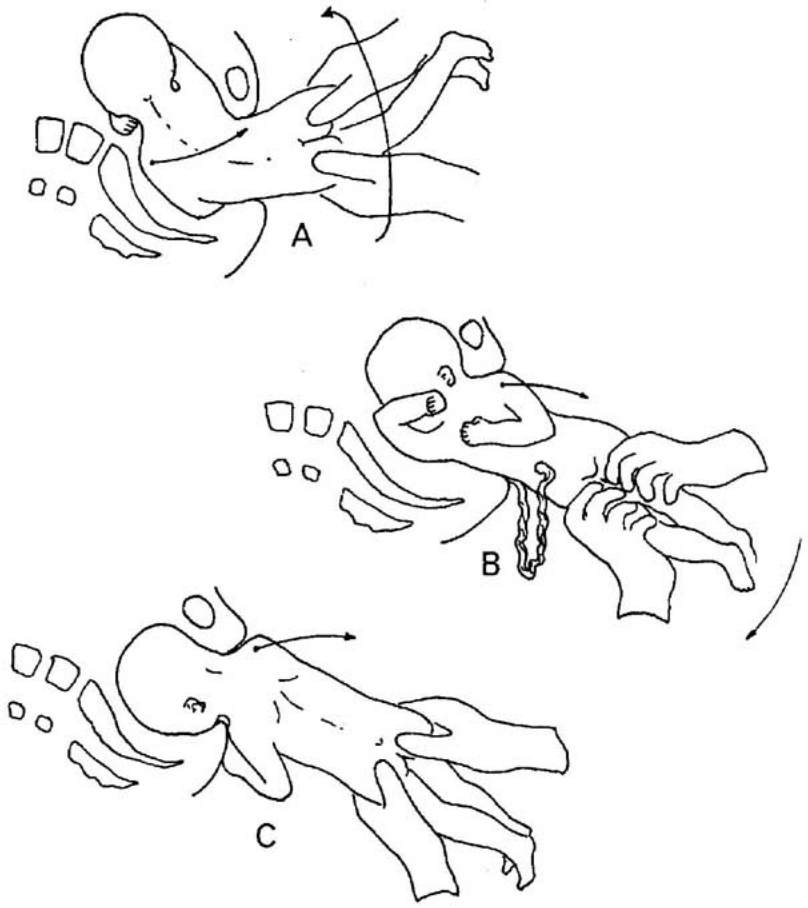

Fig. 10.11. Lovset's manoeuvre in breech with extended arm.
A. The baby's pelvis is grasped with the thumbs over the sacrum. The baby is lifted slightly to cause lateral flexion and is then rotated through 180° so that the posterior scapula becomes anterior.
B. The anterior shoulder is delivered.
C. The body has been rotated through 180°, the back being kept uppermost. Delivery of the second shoulder is now effected.

ward traction is applied. This manoeuvre is based on the fact that the pelvic inlet is so inclined that the posterior shoulder enters the pelvis before the anterior one.

i. Once the shoulders are delivered the baby is allowed to hang unsupported until the hairline is visible, fundal pressure being exerted to maintained flexion of the head (Fig. 10.12).

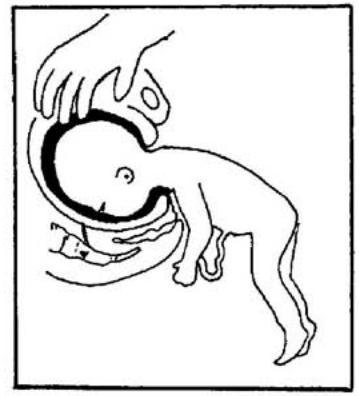

Fig. 10.12. The baby is allowed to hang unsupported. Assistant maintains flexion of the head by fundal pressure.

j. The baby's body is now lifted and forceps (e.g. Simpson's or Neville-Barnes') are applied (Fig. 10.13). If forceps are not immediately available, the Mauriceau-Smellie-Veit manoeuvre is used for the delivery of the head (Fig. 10.15).

3. **Management of Third Stage of Labour** (Breech Presentation)

This is the same as that of a cephalic presentation.

Complications of Breech Delivery

1. **Delay in Descent of the Breech:**

If delay of the breech at the brim is due to *disproportion*, Caesarean section is indicated. If it is due to *weak contractions*, a weak (1 unit per litre) oxytocin drip may be used. Arrest of the breech on the perineum is often due to extended legs. Here the anterior and then the posterior legs should be brought down by the *Pinard Manoeuvre* under anaesthesia (Fig.10.14).

Finger pressure on the baby's popliteal fossa is used to flex the knees.

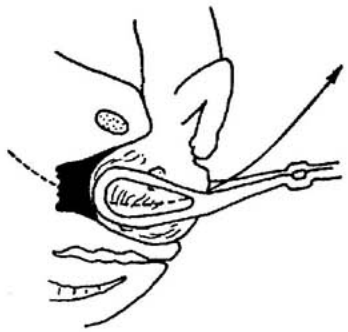

Fig. 10.13. Delivery of the head is completed using forceps.

2. **Arrest of the Arms:**

Unless traction on the buttocks was attempetd the *nuchal position* of full extension of the arms above the head, is uncommon. The Lövset technique will free most of these. The posterior arm may also be extracted in the hollow of the sacrum. In exceptional situations the humerus or the clavicle must be fractured.

3. **Arrest of the Head:**

This may be due to:

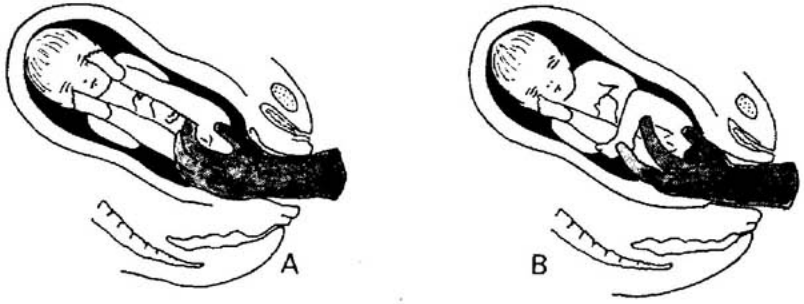

Fig. 10.14.
A. Pinard manoeuvre: first step.
B. Pinard manoeuvre: second step.

a. contracted pelvis,
b. incomplete dilatation of the cervix,
c. extended head.

Fig. 10.15. Mauriceau-Smellie-Veit manoeuvre.

If the head cannot enter the pelvis then death is usual. It is imperative that *disproportion be ruled out before labour.* Incompletely dilated cervix should be incised since waiting is not practicable.

The extended head is managed by the Mauricaeu-Smellie-Veit Manoeuvre (Fig. 10.15).

Dangers of Breech Delivery

Maternal:

1. Operative techniques increase perineal trauma.
2. Anaesthetic complications arise.

Foetal:

1. Intracranial Heamorrhage–

   Tearing of the vein of Galen may take place, due to the rapid passage of the head causing compression and decompression without time for gradual moulding.

2. Anoxia is more common due to the cord prolapse, cord depression, and aspiration of amniotic and vaginal fluid.

3. Injuries–
   These include fractures of the femur, clavicle, and the humerus, rupture of the liver, and damage to the brachial plexus.

E. Transverse Lie

Definition:

A situation in which the long axis of the foetus and the mother are at right angles to one another. Because the shoulder frequently overlies the brim this may be called the "shoulder presentation", although the back, abdomen, or ribs may also overlie the inlet. The denominator is the scapula and the site of the head determines whether the position is right or left (Fig. 10.16).

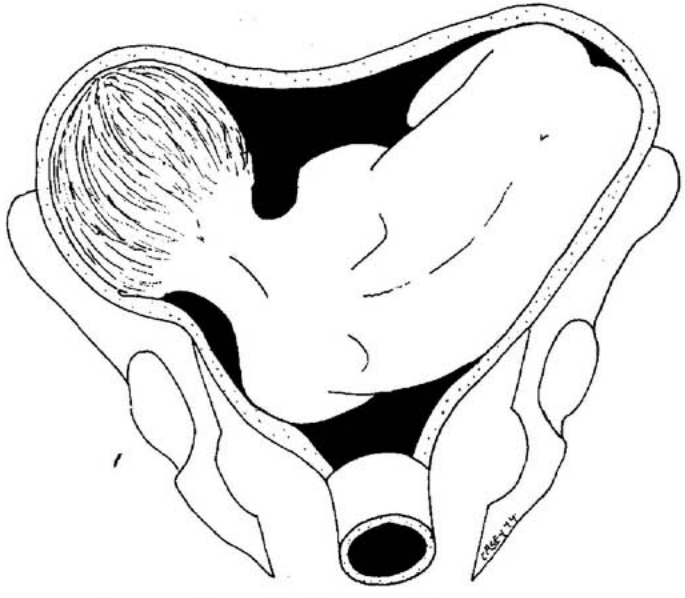

Fig. 10.16. Transverse lie or shoulder presentation.

Incidence:

Occurs in about 1/500 births.

Aetiology:

The following factors may contribute–

1. Multiparity with lax abdominal walls.
2. Anything that prevents engagement of the head, e.g. placenta praevia, fibroids, contracted pelvis.

3. Congenital abnormality of the uterus.
4. Twins and polyhydramnios.
5. Prematurity.
6. Accidental (when no cause is seen).

Diagnosis:

On abdominal palpation the head and breech are felt in opposing maternal flanks. No mass is present in the pelvis. Vaginally no foetal pole is felt – neither a head nor a breech. Sometimes a hand or shoulder may be felt.

Significance:

Transverse lie cannot deliver spontaneously as a rule, and if uncorrected, impaction, prolonged labour, infection, foetal death, and possibly uterine rupture, may claim both lives.

Management:

External cephalic version should be attempted before the onset of labour. However, the transverse lie tends to recur. Spontaneous labour should be awaited as sometimes the malposition will correct itself and a normal labour will follow.

With persistent transverse lie a Caesarean section is performed. Internal podalic version and breech extraction (a manoeuvre by which foetal polarity is changed to a breech presentation, by intra-and extra-uterine manipulations, followed by immediate, forceful delivery of the child) is dangerous and readily complicated by cord prolapse. It is therefore avoided except in those cases of second twin presentation where there is a dilated cervix and lax uterine muscle.

F. Compound Presentation

Definition:

A compound presentation is a presentation in which one or more limbs enter the pelvis with the head or the breech.

Incidence:

Occurs in less than 1/500 births. The most frequent combination is that of the head with the hand or arm.

Aetiology:

The most important factor is prematurity.

Diagnosis:

Unexplained irregularity on vaginal or rectal examination.

Management:

Usually the prolapsed part will rise out of the pelvis. If progress becomes arrested the prolapsed part should be replaced or Caesarean section considered.

G. Multiple Pregnancy

Definition:

The presence of more than one foetus in utero.

Incidence:

As an approximation–

. Twins	1:90
. Triplets	$1:90^2$
. Quadriplets	$1:90^3$
etc.	

Significance:

Twin pregnancy is associated with a perinatal mortality 2 to 3 times that of a single pregnancy. The risk to the mother is also increased. Early diagnosis and active treatment can reduce *perinatal mortality* (this is expressed as the number of stillbirths and 1st week deaths in babies of weight greater than 400 grams per 1000 births).

Varieties of Twin Pregnancies

1. **Uniovular** (Monovular, monozygotic, identical) **Twins**
   - . represents some 25% of all twins, and 40% of "like sex" twins.
   - . due to division of one fertilized ovum (polyembryony).
   - . aetiology is independent of maternal age, race, or nutritional state, but possibly hereditary plays a part.
   - . usually there is one placenta, one chorion, and two amniotic sacks (Fig. 10.17).

   - . circulations frequently intercommunicate and may result in an early death of one foetus which may become a cystic mass. A late death may lead to "mummification". At birth the transfusion syndrome may result in one foetus being anaemic and the other plethoric.

2. **Binovular** (Dizygous, Polyovular) **Twins**
   - . result from fertilization of two ova.
   - . may be of same or of different sex.
   - . more common in older age groups, especially in primigravidae.
   - . more common in Africans and Asians.
   - . chances are increased ten-fold if a previous twin pregnancy is present in maternal history.
   - . usually there are two chorions, two amnions, and two placentae (Fig. 10.17).

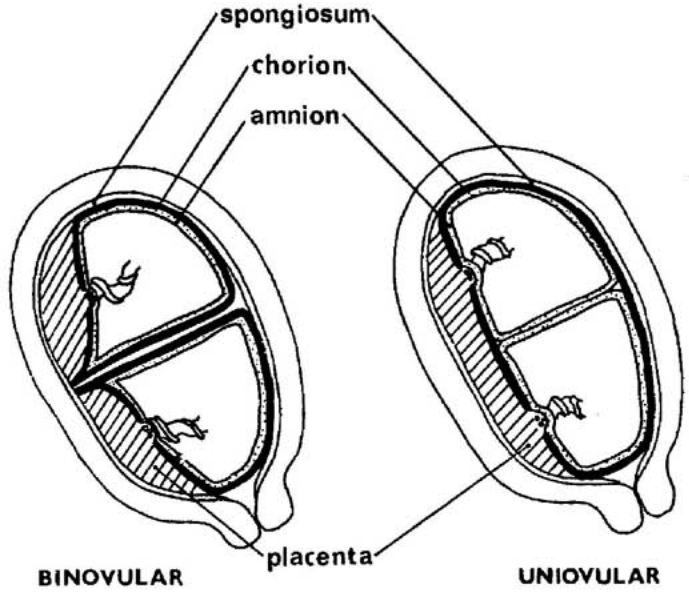

Fig. 10.17. Placenta and membranes in twin pregnancy.

Diagnosis:

1. The uterus is larger than expected at the period of amenorrhoea.

2. Palpation of three poles, preferably two heads, is possible.
3. Simultaneous auscultation of two foetal heart sounds with a difference in rate of more than 10 beats/minute. This is a difficult procedure.
4. Auscultation of two foetal heart sounds in two widely separately areas. Usually this is best performed with an utlrasonic doppler apparatus, e.g. "Doptone".
5. Ultrasonic echoscope picture of two foetuses.
6. X-ray examination must be carried out on all cases in the third trimester to confirm–
   a. the number of foetuses,
   b. foetal presentations and positions,
   c. presence or absence of conjoined twins.

When the diagnosis is made in the antenatal period (in 10% of cases it is not), differentiation must be made from –

1. Wrong dates,
2. Polyhydramnios,
3. Hydatidiform mole,
4. Large single foetus,
5. Distended bladder, ovarian cyst, uterine fibromyomata,
6. Foetal abnormality, e.g. monsters, hydrocephaly, tumours.

Complications of Multiple Pregnancy

Maternal:

1. *In Pregnancy*
   a. Discomforts of pregnancy are more severe,
   b. Anaemia is more common due to increased demands,
   c. Pre-eclampsia is 2-3 times more frequent,
   d. Hydramnios occurs in 5% of cases.
2. *In Labour*
   a. Increased frequency of operative procedures with malpresentations.
   b. Post-partum haemorrhage is more common with the large placental site.

3. *Puerperium*
   a. Heavier lochia,
   b. Slower involution,
   c. Feeding difficulties due to an increase in demand.

Foetal:

1. *In Pregnancy*
   a. Abortion is more common,
   b. Premature labour will occur in 25% of cases before week 36, and in 10%, before week 34. This prematurity is the chief cause of the high foetal wastage.
2. *In Labour*
   a. Malpresentations increase incidence of operative interference,
   b. Cord prolapse, in 90% of cases with the 2nd twin.
   c. Higher mortality rate among 2nd twins (increased by 50-100%). It is associated with delay in delivery, traumatic operative procedures, pooling of blood, prolapse of cord, and separation of own placenta.
   d. Locked twins. This is a rare complication.

Management of the Multiple Pregnancy:

Antenatal Care

1. Early diagnosis.
2. Dietary supplement –
   a. Ferrous sulphate – 320 mg b.d. (e.g. Fespan Spansule)
   b. Folic acid – 5 mg t.d.s. (e.g. Folasic)
3. Expect pre-eclampsia.
4. Additional bed rest to avoid premature labour and improve placental function. To achieve this it is best to admit the patient to hospital at 30-34 weeks and enforce rest.

Delivery

1. In hospital, by a competent operator. Anaesthetic will be required in 60% of cases.
2. Oxytocin drip must be set up at onset of labour.

3. Pudendal block is given immediately prior to delivery.
4. Deliver first twin in the same manner as a singleton.
5. Tie the umbilical cord immediately to avoid transfusion syndrome.

Second Twin –

The optimum time for delivery of the second twin is 10–15 minutes after delivery of the first twin.

1. Lie is ascertained, and if not longitudinal it is corrected by external manipulation.
2. Oxytocin drip rate is increased.
3. The second sac of membranes is ruptured as the presenting part enters the pelvis. Delivery is carried out.
4. Persistent transverse or oblique lie which cannot be corrected by external version is managed by internal podalic version and breech extraction (see page 204).

Third Stage –

1. Ergometrine 0.25 mg is given intravenously with the delivery of the second twin.
2. The oxytocin drip is kept running for four hours to gain good uterine retraction thus reducing post-partum haemorrhage.

H. Prolapse of The Umbilical Cord

Definition:

A condition where the umbilical cord lies beside or below the presenting part. The cord may occupy three positions (see Fig. 10.18).

Incidence:

Less than 1% of births.

Significance:

Although a rare complication its significance is disproportionately great because of the high foetal mortality (about 35%) associated with it.

Aetiology:

Prolapse may occur whenever the presenting part does not fit the inlet closely.

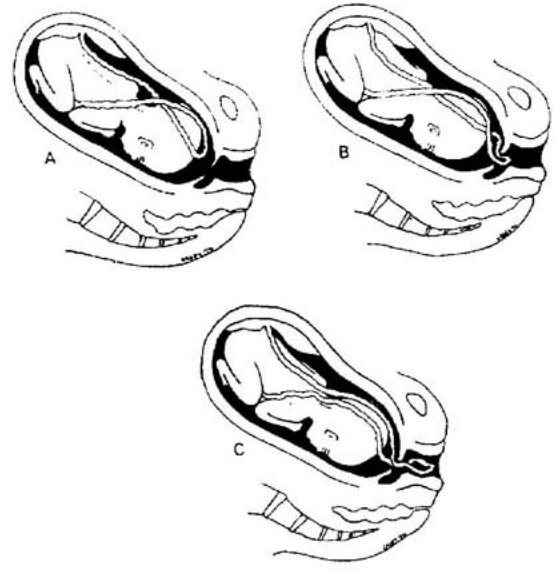

Fig. 10.18. Types of umbilical cord prolapse.
A. Cord prolapsed at the inlet.
B. Cord prolapsed into the vagina.
C. Cord prolapsed through the introitus.

1. *Abnormal Presentations* – e.g. transverse lie, breech.
2. *Prematurity* – where the presenting part is small and associated with malpresentations.
3. *Multiple pregnancy* – Polydhyramnios, malpresentation, rupture of membranes of the second twin while high in the uterus.
4. *Polyhydramnios* – washes the cord down during rupture of the membranes.
5. *High presenting part* – e.g. in disporportion.
6. *Iatrogenic* – when performing a low artificial rupture of membranes if the head is high; during disengagement, rotation or flexion of the head.

Diagnosis:

By vaginal examination.

Management:

1. If the baby is dead allow labour to progress without treatment.
2. If the baby is alive and the cervix is not in full dilatation –
   a. Hand is placed in the vagina and the presenting part is pushed away from the cord. This hand remains in the vagina until the baby is delivered operatively.
   b. At the same time the theatre is prepared for a Caesarean section.
   c. Patient is placed in the knee-chest position, or any position with hips high and head low.
   d. 100% oxygen is given to the mother.
   e. Foetal heart sounds are checked throughout transport to the theatre.
3. If the cervix is fully dilated forceps delivery is performed.
4. If difficulty with delivery is envisaged such as may occur with disproportion, or if the cervix is incompletely dilated, then a Caesarean section should be performed.

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CHAPTER 11

MATERNAL DISEASE IN PREGNANCY

General Instructional Objective

Understands the effects of maternal disease in pregnancy so that appropriate management can be instituted.

Specific Behaviours

1. Demonstrates ability to assess pregnant women with maternal disease.
2. Discusses possible effects of maternal disease on mother and foetus.
3. Discusses the principles of management of maternal disease in pregnancy.
4. Demonstrates an ability to counsel women with maternal disease.

Maternal Disease in Pregnancy

A. Anaemia

Classification:

1. *The Anaemias of Pregnancy*
   a. Iron deficient anaemia.
   This group comprises 95% of anaemias of pregnancy.
   b. The megalobalstic anaemias of pregnancy.

2. **Anaemias due to causes other than pregnancy which are found associated with pregnancy**

   a. Anaemia due to chronic renal disease

   b. Nutritional anaemias –

      . Iron deficiency
      . Folate and Vitamin B12 deficiency
      . Protein deficiency

   c. Haemolytic anaemias

   d. Aplastic anaemias

Iron Deficiency Anaemia

Iron Values

The normal adult body contains 3-5 grams of iron, distributed approximately as follows:

2,500 mgm as haemoglobin
200 mgm as myoglobin
800 – 1,200 mgm as storage iron (around 25% of total)
3 mgm as transferrin
4 mgm as cytochromes
3 mgm as catalases

Iron Absorption and Metabolism

Any ingested iron must be reduced to the ferrous form before it can be absorbed. Most of this absorption occurs through the upper small intestine. The normal daily absorption rate is about 5-10% of the ingested iron in food or about 0.6-2.0 mgm/day. The maximum possible absorption rate appears to be about 3-4 mgm/day. The actual mechanism of absorption is mediated through an active metabolic process which probably involves oxidative metabolism and copper containing enzymes. There are four factors which influence the rate of iron absorption:

1. A decrease in the size of body stores leads to an increased rate of iron absorption.

2. An increased rate of erythropoiesis leads to an increased rate of abosrption.

3. The intestinal mucosa sets an upper limit to the rate of absorption.

4. Ferritin may facilitate absorption by storing iron within the cell.

Once iron does enter the mucosal cell there is a striking stimulation of the production of apo-ferritin which combines with the iron to form ferritin.

When iron passes into the plasma it is oxidised from the ferrous (Fe ††) to the ferric (Fe †††) form and transported by a beta-l-globulin called transferrin. The blood levels of transferrin are only altered under two conditions:

1. In the case of iron deficiency the serum transferrin (i.e. serum iron binding capacity) rises from normal values (250 ug/100 ml) to levels in the vicinity of 400-500 ug/100 ml.

2. Low values may occur with disorders of protein synthesis. Normally the plasma transferrin is about 35-40% saturated, carrying between 110-125 ug/100 ml of iron.

From plasma, iron is made available to organs of storage, for haemoglobin and myoglobin synthesis, for transfer to the foetus or for excretion.

Iron is stored as ferritin and haemosiderin, this storage being mainly in the bone marrow, spleen, liver and gut mucosa.

The stores are the first fraction of body iron to be depleted if deficiency occurs.

The body at all times attempts to conserve iron and apart from loss through bile, sweat, faeces, urine and desquamated hair, nails and epithelial cells (total 0.5-1.0 mg/day) and menstrual loss (average 0.5-1.0 mg/day), there is no other mechanism by which the body normally loses iron.

When red blood cells are destroyed almost all of the iron is re-utilised by returning to the plasma pool for redistribution.

Physiology of Blood in Pregnancy

During normal pregnancies there is a fall in haemoglobin levels, from about 13 gm% (average values). This is the "physiological anaemia of pregnancy", and is explained as follows:

1. The plasma volume increases by a factor of 45%, this change beginning around the 10th week and reaching its zenith at about the 30-34th week.

2. Red cell mass also increases by about 15% (at 30-34 weeks), this being much less than the increase in plasma volume and lagging about 8-10 weeks behind it.

The increase in plasma volume over circulating red cell mass produces a physiological "anaemia". Taking this into consideration an arbitrary haemoglobin level has been taken of 11.5 gm% below which it is felt no pregnant patient should be allowed to fall without investigation.

During pregnancy there is an increase in the amount of iron metabolised, its distribution being as follows:

1.	To account for excess red cell formation	320mgm
2.	For myoglobin in the uterus	50 mgm
3.	Transferred to the foetus	200 mgm
4.	Transferred to the placenta	100 mgm

Also following pregnancy there is a loss of iron in blood (150 mg) and milk (150 mg). From these figures must be subtracted the amount of iron saved due to 9 months amenorrhoea, approximately 270 mg.

Placented Transfer of Iron

Transfer across the placental barrier probably occurs by a similar mechanism to that found in absorption from the gut. Moreover, iron is transferred against a gradient across the placenta (the foetal serum iron levels have often been found to be considerably higher than the anaemic mothers' levels).

It is probable (extrapolating from experimental results with rabbits) that the human mother ceases to utilise absorbed iron during the last 6-8 weeks of pregnancy and mobilises her own stores. This will only cause anaemia if the stores have been previously depleted.

That the majority of iron transfer to infants probably occurs during the last 6 weeks of intrauterine life is evidenced by the fact that premature infants are not only more anaemic than mature infants of the same age, but that they also absorb more iron over a given period.

The Cause of Iron Deficiency Anaemia in Pregnancy

1. Previous pregnancies may have depleted iron stores.
   Thus hypochromic anaemia is common in women who are grand multiparae.

2. Menstrual losses prior to pregnancy may have been execssive (i.e. greater than 1 mgm/day average over each month).
3. Dietary deficiency in iron may occur and foods such as cereals may interfere with absorption due to the presence of phytate, phosphates and calcium.
4. Persistent occult blood loss from the gastro-intestinal tract may lead to severe iron deficit.
5. Finally, if stores are inadequate due to any combination of the above factors, transfer of iron to the foetus, placenta and for pregnancy requirements, an iron deficiency may be unmasked.

Assessment, Diagnosis and Prevention of Iron Deficiency Anaemia in Pregnancy

History:

When a patient first presents for antenatal care, a full obstetrical history includes enquiries about previous pregnancies, past menstrual pattern, and medical and surgical diseases with particular reference to renal disorders. A specific enquiry is made about any blood loss of minor or major degree. Also it will be noted whether the patient is of a Mediterranean origin, which would indicate the possibility of Thalassaemia minor.

The symptoms of iron deficiency anaemia include lassitude, dyspnoea, palpitations, giddiness, fainting, oedema and parasthesia. Due to the peripheral vasodilatation during pregnancy, pallor is not a helpful symptom. Also subjective feelings are accepted as part of "the burden of pregnancy" and their significance may be overlooked.

Physical Examination

On physical examination, physical signs may or may not be present. These include pallor (discussed above), nail and hair changes, oedema, a raised J.V.P. and a bounding pulse (this is evidence of a hyperdynamic circulation and is often present in normal pregnancy). Because of the variability of physical signs, certain ancillary investigations are mandatory in every pregnancy.

Haemoglobin Estimation

This is carried out routinely at the first antenatal visit and again at the thirty second or thirty fourth week. If the haemoglobin

level is below acceptable limits (12.5 gm% early in pregnancy and 11.5 gm% near term), or if there is clinical evidence of iron depletion (haemoglobin may be normal when the stores have been depleted) further investigations as outlined below, must be carried out and if iron deficiency is present (as it will be in 95% of such cases), parenteral iron is indicated and should be given at least 4 weeks before the onset of labour is anticipated.

Prophylactic Oral Iron

However, if the haemoglobin level is within normal limits and there is no clinical evidence of iron depletion, an oral iron preparation is prescribed prophylactically throughout the pregnancy. Oral iron preparations are readily available and should be administered to all antenatal patients. FESPAN SPANSULE containing a slow release ferrous sulphate, may be given both antenatally and to post-natal patients.

There are many commercial preparations of iron available but recently there has been a trend towards using iron and folic acid combinations wherein the daily requirments of both compounds are administered in a simple tablet. A suitable combination is FEFOL 2 SPANSULE which contains a slow release iron compound with folic acid in sufficient amounts to provide for the requirements of a normal pregnancy. The patient should be told that her stools may appear black while she is taking the tablets. She should also be told that iron tablets are extremely dangerous if taken in large quantities and should be kept well out of reach of children.

Diagnosis of Iron Deficiency

1. Haemoblobin estimation has already been discussed.

2. Haematocrit – The P.C.V. in pregnancy is unreliable as a guide to anaemia due to physiological haemodilution.

3. Blood Smear – This is the easiest method of determining iron deficient anaemia. In iron deficient anaemia hypochromic microcytic red cells will be seen. The other causes of this picture are Thalasseamia minor (Thalassaemia major is diagnosed clinically), other haemoglobinopathies, chronic disease processes and sideroblastic anaemia.

4. Indices – These are of particular value, giving a quantitative assessment of what one visualises on a blood smear.

   a. Mean Corpusular Volume (haematocrit divided by the red cell count). The normal value is 86.7 cubic microns. A decrease in this occurs early in iron deficient anaemia.

   b. Mean Corpusular Haemoglobin (total haemolgobin in gms% divided by the red cell count). The average normal value is 29 micrograms. This also decreases early in iron deficient anaemia.

   c. Mean Corpusular Haemoglobin Concentration (haemoglobin in gms% divided by the haematocrit). The average normal value is 33.6%. This decreases later in the course of iron deficiency anaemia, when the M.C.H. is falling at a faster rate than the M.C.V.

5. Serum Iron and Serum Iron Binding Capacity – In iron deficiency anaemia serum iron levels will generally be reduced below 60μgm/100 ml and the iron binding capacity will rise above 450μgm/100 ml.

6. Marrow Aspiration – Staining aspirate with Pearl's stain will allow visualisation of iron stores and is the definitive method of diagnosing iron deficiency as opposed to other causes of hypochromic, microcytic anaemia. It can also reveal iron depletion prior to a fall in haemoglobin.

Complications of Iron Deficiency Anaemia

Maternal:

1. *Shock*

   This occurs more quickly in anaemic patients when blood loss or some other cause of shock occurs.

2. *Infection*

   This occurs more commonly in anaemic women and is more difficult to cure.

3. *Heart Failure*

   The risk is increased in anaemia because of the secondary increase in cardiac output.

4. *Premature Labour*
   Occurs more commonly in anaemic patients.

5. *Hypertensive and oedematous states*

Also occur more frequently in anaemic patients.

Foetal:

Foetal complications are mainly secondary to maternal complications, the foetus extracting enough maternal iron for its own needs at the expense of the mother.

Treatment of Iron Deficiency Anaemia

1. Routine prophylaxis – already discussed.
2. If hypochromic microcytic anaemia occurs then the patient will be unable to absorb enough iron orally to restore normal levels. In such a case parenteral administration is indicated.

   Oral Iron Preparations

   Ferrous sulphate compounds are the most commonly used in all preparations and 90% of patients tolerate this well (FEFOL 2 contains 270 mgm of ferrous sulphate and 300 micrograms of folic acid').

   FESPAN containing ferrous sulphate can be administered over a prolonged period of time for antenatal and post-natal patients.

   Alternative oral preparations which may be used if the patient is sensitive to ferrous sulphate are:

   . Ferrous gluconate
   . Ferrous fumerate
   . Ferrous carbonate
   . Ferrous succinate
   . Ferric ammonium cirtate

   If the prophylaxis fails and the patient becomes anaemic it may be because:

   a. Patient failure to take iron due to :
      i. Gastric upsets,
      ii. Laziness.
   b. Failure of absorption due to achlorhydria, presence of non reduced iron salts, or intestinal malabsorption.
   c. Failure of transport of iron (rare).

   d. Failure of utilisation (sideroblastic anaemia).

Parenteral Iron

1. *Iron Dextran* (Imferon) – Total dose infusion.

   This may be given as a single dose I.V. infusion with excellent results.

   For each 0.7gm% of haemoglobin below normal values give 100 mgm. of parenteral iron plus 500 mgm. to replenish stores.

   Imferon contains 50 mgm of iron per ml. So, for example, 20 mls. Imferon are required if 1,000 mgm or iron is to be administered. The infusion should be given in one litre of normal saline with a maximum concentration of 5%.

   Side effects are minimal and the total infusion can be given over 2-3 hours.

   There is an average rise in haemoglobin of 1.0gm% for each week of the first month of treatment, after an initial lag during the first week.

2. *Iron Dextran* – single dose injections.

   Dose: Single shot injections of 5 ml or 10 ml I.V. The injection is given over 2-3 mins. and may be repeated some weeks later. Other methods of parenteral administration of iron are less satisfactory.

   Note: Anaemic patients should never be transfused with blood unless bleeding or with impending congestive cardiac failure when they may require exchange transfusion. Always have blood cross-matched for anaemic patients in labour but do not transfuse unless haemorrhage occurs.

Megaloblastic Anaemia of Pregnancy

In pregnancy this is usually due to folic acid deficiency.

Folic Acid Metabolism:

Folic acid, a combination of the pteridine nucleus, p-aminobenzoic acid and glutamic acid, occurs naturally in green-leaf vegetables. Deficiency may occur whenever the diet is deficient or more commonly when erythropoiesis creates a greater demand for folic acid.

Folic acid is the basis of a particular group of co-enzymes participating in the synthesis of purines and thymine in the formation of nucleic acids. Folinic acid, a folic acid coenzyme is necessary for the metabolic degradation of histidine, and absence leads to accumulation of formimoglutamic acid (FIGLU). This can be estimated in urine and used as a parameter of folic acid deficiency.

Assessment and Diagnosis of Folic Acid Deficiency in Pregnancy: Folic acid deficiency is suspected if the haemoglobin value in a pregnant patient falls below 9 gm%. In such a case bone narrow examination should be performed and in megaloblastic anaemia will reveal megaloblasts. Blood smear or blood indices reveal a macrocytic anaemia with hypochromia only if there is concomittant iron deficiency. Symptoms include pallor, fatigue, faintness and occasionally jaundice. Vomiting may occur and occasionally the patient will have glossitis and splenic enlargement. If the disorder progresses untreated during pregnancy the haemoglobin may drop as low as 3-4 gm% especially after the 34th week. This would commonly lead to congestive cardiac failure and death.

Treatment:

Give 5 mgm Folic acid/day orally.

Anaemia due to Chronic Renal Disease

The aneamia is thought to be due to a reduction in the production of erythropoietin by the kidney.

The signs and symptoms of iron deficiency anaemia may be present and the haemoglobin will be depressed. Blood smear or determination of indices discloses a normocytic, normochromic anaemia with no excess megaloblasts.

Iron and folate therapy do not effect the disease. The renal tract in such cases should be investigated thoroughly, including an I.V.P. if necessary and any treatable disorder such as chronic infection should be treated vigorously.

B. Urinary Tract Injection

Introduction

Disease of the kidneys, of which pyelonephritis is the most important are responsible for 5-10% of all deaths. Moreover, the incidence of pyelonephritis is increased in pregnancy and it has

very deleterious effects on the foetus and mother. Consequently, urinary tract infection must be excluded in every pregnancy and, if present, treated vigorously.

Increased Incidence of Urinary Tract Infection in Pregnancy

Women, because of their relatively short, straight urethra, are particularly prone to contract urinary tract infections and certain physiological changes in pregnancy increase the risk:

1. Placental steroids lead to relaxation of the smooth muscle of the bladder, ureters, and calyceal systems (as well as smooth muscle elsewhere) with a resulting increase in capacity. (Up to 200 ml each in the case of the ureters). This predisposes to urinary stasis and vesico-ureteric reflux with consequent growth and dissemination of organisms.
2. The growing uterus begins encroaching on bladder capacity at about 8 weeks amenorrhoea. This is associated with elongation and funnelling of the urethra. Apart from causing frequency of micturition, this may cause a failure to completely empty the bladder, also predisposing to growth and spread of organisms.

Effect of Urinary Tract Infection on Pregnancy

1. *Effect on Foetus*

   It is thought that toxins produced by renal infection (e.g. endotoxins produced by gram-negative organisms) produce fibrinoid changes within arterioles and capillaries, especially those supplying the deciduo-placental area. This leads to reduced blood flow and an increased incidence of antepartum haemorrhage, premature labour, intrauterine death and neonatal death.

2. *Effect on mother*

   Pregnancy causes a deterioration of the condition of mothers with chronic renal disease. 25-30% of mothers with elevated blood urea will die within 12 moths and a further 30% will require renal transplants. Also, the complications of urinary tract infection, especially ante-partum haemorrhage and intra-uterine death may endanger the mother's life.

Prognosis and Assessment of Urinary Tract Infection in Pregnancy

1. *History:*

   If a full obstetrical history is taken then the presence of symptoms relevant to urinary tract infection will be ascertained. Frequency occurs in the early and late stages of pregnancy without infection but scalding on micturition, and suprapubic pain are abnormal. The full obstetrical history also includes enquiry into past urinary tract infections. The frequency and severity of these and the adequacy of treatment should be noted. The occurrence of accidental haemorrhage, premature, labour, intrauterine death or perinatal death in previous pregnancies will arouse immediate suspicion of chronic renal infection.

2. *Examination:*

   Little difficulty will be encountered in the diagnosis of acute pyelonephritis but physical examination is often of little help in diagnosing chronic renal disease, and is of no help if the disease is subclinical.

3. *Examination of Urine:*

   The most convenient method of collecting urine for bacteriological diagnosis is to obtain a mid-stream specimen. To avoid contamination, the vulva and vagina are first swabbed with cotton wool swabs dipped in saline or tap water (no antiseptics are used) then dried thoroughly. The patient is then instructed to collect the mid-portion of the stream in a sterile collecting jar. Although 95% of such specimens give accurate results, any positive cultures must be repeated for confirmation.

   Collection of a specimen by catheterisation is a time consuming procedure, which, as a sterile procedure, carries a 10% risk of infecting the bladder.

   Suprapubic aspiration is quick and convenient but care must be taken to avoid damaging the foetus.

   a. *Micro-urine examination*

      A portion of the specimen is centrifuged, and the sediment is examined microscopically for the presence of white cells, red cells, epithelial cells, casts, mucous and bacteria.

   b. *Culture*

      Culturing must be performed immediately or alternately the urine must be stored at 4°C during the intervening interval (less than 12 hours).

   The culturing methods used are:

   i. *Bacilluria Screening Test* (B.S.T.)
   Every pregnant woman must have this done at her first visit. A strip of blotting paper 3" x $\frac{1}{4}$" is bent over $\frac{1}{2}$" from one end and dipped in urine, soaking up a standard volume. The $\frac{1}{4}$" x $\frac{1}{2}$" portion is then applied to an agar slope causing any organisms on it to be transferred to the agar. This is cultured overnight. The subsequent growth of over 25 colonies of one organism is indicative of an organism count of greater than 100,000 per ml. which is regarded as evidence of a urinary tract infection until proven otherwise.

      The organisms most commonly cultured are E. coli, Strep. faecalis, Proteus and Klebsiella.

   ii. *Pour-plate Dilution Technique*

      This is used if the B.S.T. produces a positive result, or if urinary tract infection is suspected on clinical grounds and antibiotic sensitivities are to be determined. Two blood agar plates are prepared. Onto the first is poured 1 ml. of urine. Onto the second is poured 1 ml. of urine diluted 100 times. This method permits a quantitative estimation of the number of organisms present. If the growth is not pure (i.e. there is more than one type of organism growing profusely), then contamination must be assumed and a fresh specimen taken for culture.

4. *Leucocyte Excretion Rate* (L.E.R.)

   The presence of an excessive number of white blood cells in a urine specimen usually indicates urinary tract infection. If the numbers of white cells excreted in urine are counted (number in a measured volume x total volume in unit time). it is found that in a normal, non-pregnant woman 200,000-400,000 per hour, or less, are excreted. In pregnancy, due to

the increased renal blood flow the normal acceptable value is from 400,000 to 1000,000 per hour.

The L.E.R. is particularly useful in assessing asymptomatic bacilluria in which a high L.E.R. indicates the probability of an underlying pathological process as opposed to simple bladder bacteruria.

5. *Measurement of Blood Urea and Serum Creatinine*

These measurements are particularly used to assess renal function when damage to kidney parenchyma is suspected as in chronic or subclinical pyelonehpritis. The levels are lowered in pregancy due to the increased glomerular filtration rate and renal clearance, the upper acceptable limits being:

. blood urea – 28 mgm/100 ml.
. serum creatinine – 2 mgm/100 ml.

The creatinine clearance is increased in pregnancy so that normal values rise from 80-100 mgm/minute in the non-pregnant woman, to 120-150 ml/minute in the pregnant patient.

Forms of Urinary Tract Infection and their significance

1. *Asymptomatic Bacilluria* (Sub-clinical urinary tract infection) This is probably twice as common as symptomatic urinary tract infection and is the presence of more than 100,000 orgs./ml in urine with no symptoms of urinary tract infection. Pathologically it is of three types:

   a. Organisms multiplying in the urine but not in the tissue of the bladder or kidney.

      . Symptoms and signs: Absent.
      . Microurine: Will show white cells, epithelial cells, scanty red cells and bacteria. L.E.R. usually normal.
      . Effect on pregnancy: No effect, but predisposes to tissue infection.

   b. Invasion of the bladder wall, usually over the trigone, by organisms (sub-clinical cystitis).
      . Symptoms and signs: Often not recognised. (Frequency is misinterpreted as a normal symptom of pregnancy and dysuria is absent due to lack of urethral involvement.)

      . Microurine: As for (a.) but with more cells, mucous and debris present. L.E.R. usually elevated.
      . Effect on pregnancy: No effect, but predisposes to pyelonephritis.

   c. Subclinical pyelonephritis. (This comprises 50% of cases of asymptomatic bacilluria).

      . Symptoms and signs: Either absent or not recongised as abnormal until viewed retrospectively, e.g. chronic tiredness and malaise.
      . Microurine: As for (b.) with the addition of casts-hyaline, granular and to a lesser extent, red cell. L.E.R. usually raised.
      . Effects on pregnancy: In cases with a raised L.E.R. the incidence of premature labour is 20%; of ante-partum haemorrhage 12%; and of peri-natal death 5%.

2. *Cystitis*

This is infection localised to bladder tissue.
. Symptoms and signs: Frequency and dysuria. Usually there is no generalised toxic reaction due to the resistence of the bladder wall to infection and penetration of toxin.
. Microurine: As for subclinical cystitis. L.E.R. raised.
. Effect on pregnancy: No effect but predisposes to pyelonephritis.

3. *Pyelonephritis*

   a. *Acute*

      . Symptoms and signs:
      Acute systemic illness (due to bacterial toxins) with pyrexia, malaise and possibly vomiting and dehydration. Loin pain and tenderness are usually present.
      . Microurine:
      Granular and red cell casts will generally be present in addition to the findings in cystitis.
      . Effects on pregnancy:
      Pyrexia causes 25% to go into premature labour. Accidental haemorrhage occurs in 15%. Perinatal loss from intrauterine death, growth retardation, or prematurity occurs in 5-10%.

b. *Chronic*

This is characterised by chronic interstitial fibrosis with gross destruction of renal parenchyma.

. Symptoms and signs:
These are often minimal. Chronic tiredness and malaise may be present. Acute exacerbations may occur with the symptoms of acute pyelonephritis. Alternatively, the patient may present with complications such as hypertension or pre-eclampsia.

. Microuine:
Casts (granular or red cell) confirm the diagnosis. The presence of red blood cells in sufficient quantity to be termed haematuria is an indication for I.V.P., an investigation which is otherwise usually postponed until after delivery.

. Effects on pregnancy:
Chronic pyelonephritis is associated with hypertension and pre-eclampsia (with all of their complications) in up to 75% of patients.
Foetal loss occurs in 35%. Any woman with a blood urea of greater than 50 mg/100 ml in pregnancy runs 50% risk of losing her infant. Deterioration of renal function occurs in 50% of mothers with chronic pyelonephritis.

Management of Urinary Tract Infection in Pregnancy

1. *Asymptomatic Bacilluria*

   Determine L.E.R. if this is elevated, treat as for chronic pyelonephritis (see below). If it is low, a short course of the appropriate antibiotic should eliminate the organisms. Repeat urine samples should be taken to ensure this.

2. *Cystitis*

   a. Take a contaminant free urine specimen for microscopic examination, culture and antibiotic sensitivity.

   b. Institute appropriate chemotherapy. Use Furadantin, Gantrisin, Bactrim initially. Alternatives are ampicillin, nalidixic acid, streptomycin, kanamycin and methenamine, among others (see end of this chapter for details of drugs).

      Chemotherapy may be altered according to the response of the patient or the results of sensitivity tests. Continue treatment for 10 days. Re-examine urine at 14 days and again at 6 weeks.

3. *Acute Pyelonephritis*

   a. Admit to hospital.

   b. Obtain a contaminant – free specimen of urine and institute antibiotic therapy as for cystitis. Bactrim is probably the best chemotherapeutic agent initially and the infection then is kept suppressed with Furadantin.

   c. Re-hydrate the patient.

   d. Give analgesics if necessary.

4. *Chronic Pyelonephritis*

   In this case the culturing of organism and determination of antibiotic sensitively is most important and should be carried out accurately before initiation of treatment.

   Introduce appropriate chemotherapy and rotate the agents, e.g. Bactrim-nalidixic acid, Furadantin-ampicillin.

   Treatment should continue for at least one month or until organisms can no longer be isolated from the urine.

Drugs used in Urinary Tract Chemotherapy

Bactrim:

Trimethoprim 80 mg. sulphamethoxazole 400 mg per tablet.
Dose: 2 tabs. bd. after meals.

Action:

Sequential blockage of two enzymes acting within the bacterial metabolic pathway of folinic acid synthesis.

The combination is bacteriocidal at concentrations where the individual components are bacteriostatic.

Side effects:

1. Nausea, vomiting, glossitis (rare).

2. Sulphonamide sensitivity causing rashes.
3. Haematalogical change (e.g. leukopenia, thrombocytopenia) – especially if used over a long term.

Contra-indications:

1. Marked liver parenchynal damage.
2. Blood dyscrasias.
3. Severe renal insufficiency.
4. Folate deficiency.

Furadantin:

Dose: 200-400 mg per day in divided doses orally.

Reaches high levels when concentrated in urine and is therefore excellent for treating infection in the urinary pathway. e.g. cystitis, and for maintaining the urine free of organisms.

Side effects:

1. Nausea and vomiting (common).
2. Skin rashes.

Ampicillin:

Dose: 500 mgm 6 hourly.

Action:

Interferes with the production of the cell wall of certain gram positive and gram negative bacteria.

Side effects:

1. Hypersensitivity – rashes are common.
2. Convulsions if toxic levels (rare).
3. Superinfection of bowel if used in very high doses.

Streptomycin:

Dose: 1-2 gm per day I.M.

Action:

Interferes with bacterial protein synthesis.

Side effects:

1. Toxic to vestibular division of the eight cranial nerve, (especially if in doses over 1 gm per day for longer than one month).
2. Allergic reactions.

Contraindication:
Impairment of renal function (may be used with caution if blood levels are monitored).

Mandelamine:

Dose: 500-1000 mg per day in divided doses.

Action:

Liberates formaldehyde in acid urine (if PH below 6.0) – may need to give NH4Cl 0.5–1.0 gm t.d.s. to acidify urine.

Side effects:

Very few. May get gastric irritation.

C. Diabetes Mellitus

The significances of diabetes mellitus in pregnancy is related to the high incidence of maternal complications and the grossly elevated perinatal mortality rate.

Definitions:

1. *Clinical Diabetes* – diabetic response to a G.T.T. with the symtoms or complications of diabetes.
2. *Chemical Diabetes* – a diabetic response to a G.T.T. without clinical abnormalities.
3. *Latent Diabetes* – a condition in which there is a normal G.T.T. currently, but there has been either a diabetic G.T.T. or abnormal blood glucose response to provocative tests.
4. *Potential Diabetes* – a condition in which there is a normal G.T.T. but there is an increased risk of developing diabetes because the patient:
   a. is an identical twin, the other twin being diabetic,
   b. has both parents diabetic,
   c. has one diabetic parent, and the other non-diabetic parent

has either a diabetic parent, sibling or offspring, or a sibling with a diabetic child,

d. has borne a live or stillborn child weighing 10 lb. (4.5 kg) or more at birth, or a stillborn child showing hyperplasia of the pancreatic islets, not associated with Rhesus incompatibility.

5. *Pre-diabetes* – that period of the life of a diabetic before the diagnosis is made. This can only be determined in retrospect.

Effects of Pregnancy on Glucose Metabolism

1. **Human Placental Lactogen**

   This hormone has an anti-insulin and a lipolytic effect leading to reduced glucose tolerance. The result is that blood glucose levels rise, making more glucose available to the foetus.

   Normally, this is largely compensated for by an increased production of maternal insulin but in some women it may lead to "gestational diabetes". Human placental lactogen differs from human growth hormone with respect to its effect on carbohydrate metabolism in that there is no negative feedback by high blood glucose levels on the centres of production.

2. Other steriods produced by the placenta (oestrogens and progestogens) also probably have an insulin-antagonistic effect.

3. The placenta destroys insulin.

4. Glomerular filtration rate is increased during pregnancy (reflecting the 50% increase in renal blood flow) and thus more glucose is filtered through the glomerular capillaries. However, there is no corresponding increase in the tubular resorption mechanism for glucose so glucose may be excreted in the urine of an otherwise normal, pregnant woman. In a non-diabetic woman this glycosuria is harmless but in a diabetic it may result in dangerous loss of carbohydrate, difficulty in control and an increased tendency to ketosis.

Incidence of Diabetes Mellitus with respect to Pregnancy

3% of the population suffer from diabetes mellitus and about 1/6 of these are women of child bearing age. The fertility rate in diabetic women has risen from about 2% to 25-30% with the improvements in treatment made possible by insulin so that now about 1/400 pregnancies will be complicated by diabetes.

Assessment and Diagnosis of Diabetes in Pregnancy

1. **History:**

   A pregnant woman's history may indicate the risk of potential diabetes (see Definition). If a patient actually develops diabetes in pregnancy this will normally be diagnosed long before symptoms occur following routine urinalysis, but if this is not so it is possible that symptoms of polydipsia, polyuria, polyphagia, or severe pruritis valvae will lead to the diagnosis. Even more rarely, the patient may present acutely in a state of hyperglycaemia and keto-acidosis.

2. **Physical Examination:**

   If diabetes mellitus is present or is suspected, examination will include all systems, particular attention being paid to the C.V.S., C.N.S., optic fundi and the possibility of infection.

3. **Investigation:**

   a. Routine urinalysis, including a specific test for glucose, is performed at every antenatal visit. A positive result may merely be due to the normal physiological changes of pregnancy but in order not to miss any cases, it must be assumed that the patient with glycosuria has diabetes mellitus until proved otherwise.

   b. *Glucose Tolerance Test* (G.T.T.)

      The patient has a normal carbohydrate rich diet for three days then fasts for 8 hours before the test. A loading dose of 40 gm of glucose per square meter of body surface then given. (Intravenous administration is preferable because of the variability in gastric emptying time and glucose absorption during pregnancy). Diabetes mellitus is diagnosed if two or more of the following blood glucose levels are reached.

   i. Fasting level of 90 mgm%
   ii. 1 hours level of 165 mgm%
   iii. 2 hours level of 145 mgm%
   iv. 3 hours level of 125 mgm%

Effects of Diabetes Mellitus on the Mother and Baby

The incidence of maternal complications is related directly to the adequacy at control of the disease. The foetal loss is related to the

incidence of maternal complications. With proper antenatal care maternal mortality should not be greater than 0.2%. Foetal mortality on the other hand ranges from 10–30%, slightly more than half of those being stillborn, the remainder dying in the neonatal period.

1. **Effects on the Mother**

   a. *The first and second trimester:*

      i. Infections are more common. Severe moniliasis is often present and pruritis vulvae may be the first symptom of diabetes. Urinary tract infection is more likely to occur, the rate of bacteruria being 18% (three times higher than when diabetes is not present). Infection may lead to insulin resistance and keto-acidosis.

      ii. Higher rate of abortion.

      iii. Nausea and vomiting may lead, on the one hand, to insulin shock in women who are receiving insulin, and, on the other, to insulin resistance if the starvation is severe enough to cause ketosis.

   b. *Third Trimester:*

      i. Pre-eclampsia – the incidence is increased from 2 to 4 times. The perinatal loss is doubled in diabetics with this complication as compared to diabetics who do not develop it.

      ii. Polyhydramnios – the incidence in diabetes is 20-50%. A reduction in liquor can be achieved with strict control of maternal diabetes. Although the cause of hydramanios in diabetes is not known it more frequently accompanies profound metabloic disturbance than congenital malformations. It may lead to premature rupture of the membranes.

      iii. Dystocia – This may occur because of the increased incidence of large babies.

2. **Effects on the Baby**

   Maternal insulin does not cross the placenta but maternal glucose does. The foetal islet cells mature at an early age in pregnancy and if the mother is diabetic the foetal islets undergo hyperplasia as a response to the elevated blood sugar levels. The resultant increased foetal insulin is though to have two significant effects:

   a. If has a growth hormone-like effect resulting in macrosomia of all foetal organs except the brain. The foetus is thus larger than others of similar gestational age.

   b. When maternal blood glucose falls in response to exogenous insulin administration, the foetal insulin (half life to 2-4 hours), does not fall rapidly enough from its elevated level to prevent the foetus suffering hypoglycaemia. It is thought that repeated episodes such as this contribute to foetal morbidity and mortality.

   Note: Severe maternal diabetes may result in the baby being smaller than average size, rather than larger.

Intrauterine Complications

1. **Foetal abnormalities**

   The incidence in diabetes is approximately 6%. Although these abnormalities are often not severe, maternal diabetes may in some instances lead to neurologic and psychologic deficits in the child. It has been reported that children whose diabetic mothers develop ketosis during pregnancy have a lower I.Q. than do the offspring of diabetic women without acetonuria.

2. **Intrauterine death**

   The incidence of this increases markedly from the 34th week. Many of the deaths are associated with pre-eclampsia, maternal ketosis, hydramnios, and probably repetitive foetal hypoglycaemia.

Neonatal Complications

1. **Respiratory distress syndrome**

   The incidence of hyaline membrane disease is increased for two reasons. Firstly, premature delivery is more common and, secondly, in the "large baby" type of diabetes, surfactant production in the lungs is delayed. (This may be evaluated before delivery by recording lecithin/sphingomyelin ratios in the amniotic fluid.)

2. **Hypoglycaemia**

   Increased blood insulin secondary to islet cell hypertrophy may result in neonatal hypoglycaemia if administration of glucose rich fluids is delayed. Prematurity makes this complication even more likely.

3. **Birth trauma and cord prolapse**

These complications are the result of the large size of the baby and the increased incidence of hydramnios.

The Management of Diabetes in Pregnancy

This should be carried out exclusively by teams of specialists familiar with the medical, obstetrical and paediatric problems which may arise.

Two important facts should constantly be remembered:

1. Insulin requirements increase during pregnancy.
2. A lowered renal threshold occurs. Thus urinary testing as a means of assessing control is very unreliable except for the detection of ketones.

At the first visit the diabetic patient should be admitted for stabilisation and investigation. She should then be seen at least every 2 weeks until 30 weeks. Accurate assessments should be made early of retinal changes, renal function (creatinine clearance, blood urea), skin lesions and other complications. Also fasting lipids and insulin levels should be determined.

Accurate clinical evaluation of the stage of gestation is of utmost importance and this is aided by an echogram at 20-24 weeks.

Patients should be admitted to hospital at the first sign of ketosis, hypertension or hydramnios. All patients receiving insulin should be admitted at approximately the 30th week until delivery. Once in hospital the patient remains in bed for 20 hours daily. Repeated blood sugar estimation are made and the 2 hour post-prandial B.S.L. should be kept in the range of 90-120 mgm%.

Diet – this should contain no more than 250 mgm carbohydrate daily. Protein is allowed at the rate of 26 gm per kg. The remainder of the caloric intake is provided by fat to allow a caloric intake of 20 per kg per day.

Insulin – if insulin is to be used to control the disease it is best to use a soluble insulin. The dose is adjusted according to the 2 hour post-prandial B.S.L.'s. These are performed at least 3 times weekly in hospital and at each clinic visit.

Oral hypoglycaemics – may only be used if good control can be achieved and the diabetes is mild in nature. In high concentration they may have a teratogenic effect.

Time of delivery

The presence of deteriorating P.E.T., increasing hydramnios or frequent attacks of ketosis may make termination obligatory as early as the 32nd or 34th week. Most diabetics with good control, may be permitted to continue the pregnancy to the 37th week. This time is chosen because intrauterine foetal death is common after this time. However, a separate decision should be made for each patient after considering all factors. Never allow the pregnancy to exceed 38/52 if gestational age is accurately known.

Method of delivery

If the head is engaged and the cervix ripe, amniotomy is performed and an oxytocin infusion commenced at about 8.00 a.m. If labour is not well established within 4 hours then Caesarean Section is performed.

During labour, calories are provided via a dextrose drip and insulin dosage is determined by estimation of the B.S.L.

Puerperium

Soon after delivery, the insulin requirements of the mother fall, and care should be taken to avoid hypoglycaemia.

There is a greater risk of puerperal infection and breast feeding is often unsuccessful.

D. Cardiac Disease

Incidence

Heart disease is estimated to occur in about 1% of pregnant mothers.

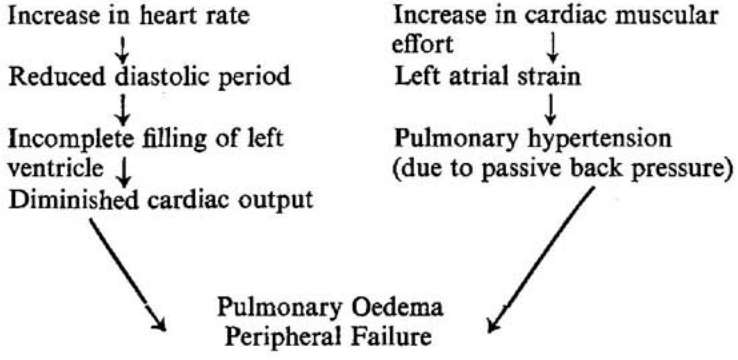
Aetiology

1. Rheumatic heart disease – Mitral stenosis is the most prevalent cardiac lesion in pregnancy.
2. Congenital heart disease – with better prophylaxis of rheumatic heart disease the incidence of C.H.D. as a complication of pregnancy is rising.
3. Cardiac disease secondary to hypertension – Relatively less common.

4. Cardiac disease associated with coronary artery disease, thyroid disorders, syphilis, kyphoscoliosis, cor pulmonale, constrictive pericarditis, heart block, myocarditis and other conditions are rare as complications of pregnancy.

Haemodynamic Changes Occurring in Heart Disease during Pregnancy
(using mitral stenosis as an example)

In mitral stenosis, the reduced size of the mitral orifice produces resistence to the increased blood flow of pregnancy, proportional to the fourth power of the decrease in radius. The heart attempts to compensate for this by increasing heart rate and muscular effort. The results are summarised in the following diagram.



The transition to cardiac failure (failure of the heart to maintain a cardiac output sufficient to meet the demands of the body tissues) may occur gradually during pregnancy or acute failure may occur in early pregnancy when the blood volume begins to increase. Most commonly, however, acute failure is precipitated by some change which induces tachycardia resulting in reduced diastolic filling and thus reduced cardiac output. The most important precipitating factors are:

1. *Anaemia* –

   Reduces oxygen carrying capacity of the blood and produces a demand for increased cardiac output. The response is tachycardia.

2. *Respiratory infection* –

   Reduces blood oxygenation and causes fever, both of which are associated with tachycardia.

3. *Any febrile illness* –

   Produces tachycardia.

4. *Exercise* –

   Produces tachycardia.

5. The sudden *increase in blood volume* immediately after the third stage of labour may precipitate failure.

Assessment and Diagnosis:

Usually a patient with cardiac disease will already be known to have the condition but occasionally a cardiac lesion will not be picked up until pregnancy both exposes the patient to physical examination, and creates increased demands on her heart.

When heart failure does occur in pregnancy it is nearly always a left heart failure, with the forward component of failure predominating. The common symptoms are limitation of activity, fatigue, palpitations, sometimes pain of an anginal type, and dyspnoea, depending on the degree of pulmonary congestion.

If right heart failure, with cyanosis, raised J.V.P. enlarged, tender liver and cardiac oedema, does occur, it usually progresses gradually and its response to bed rest, digitalis and Cardophyllin is good. In assessing a cardiac lesion, its grading, rather than the anatomical lesion, is more important with respect to management.

- Grade 1 – The patient has no symptoms and no limitation of physical activity.
- Grade 2 – The patient is comfortable at rest. Routine physical activity causes fatigue, palpitations and dyspnoea.
- Grade 3 – The patient is comfortable at rest, but even slight activity causes palpitations, dyspnoea and possibly anginal pain.
- Grade 4 – Even when resting, the patient is dyspnoeic.

Regular assessment throughout pregnancy is essential. On each occasion the patient's exercise tolerance must be accurately estimated and a full examination of the cardiovascular system must be performed, followed by E.C.G. evaluation. Also, a constant careful watch must be maintained to prevent excessive weight gain, anaemia or infection.

Management:

Grades 1 to 2

Advise daily rest periods and see the patient frequently (every 2 weeks

if possible) Ensure good dental hygiene (to prevent subacute bacterial endocarditis), treat any febrile illness aggressively and watch for the development of anaemia or excessive weight gain.

Hospitalise at 28 weeks for 2–14 days. Re-assess during this time then see the patient every 2 weeks thereafter. Hospitalise at 36 weeks until delivery. This may prevent premature labour and will ensure good rest prior to the stresses of labour and the puerperium.

If at any time during the pregnancy early signs of cardiac failure develop then:

1. Admit to hospital for complete bed rest. The administration of a sedative will make this more bearable for the patient and will help reduce the stress of anxiety. Valium 2-5 mg t.d.s. is useful in this respect.
2. Digoxin may be given with care.
3. Restrict fluid intake and give diuretics.

Grades 3 to 4

Hospitalise for the duration of the pregnancy. Patients with Grade 4 disability should be totally confined to bed.

If pulmonary oedema occurs (main features are intense dyspnoea with bronchospasm), treat urgently as follows:

1. Raise the head of the bed.
2. Administer Morphine 15 mg I.V. This relieves anxiety and may directly contribute to relief of bronchospasm.
3. Give oxygen by face mask until an oxygen tent is available.
4. Relieve bronchospasm. Administer aminophyllin 250 mgm I.V. over 10 minutes.
5. Reduce fluid volume:–
   a. Administer Lasix 40 mg I.V. stat.
   b. Apply torniques to three limbs and rotate every 10 minutes.
   c. Alternatively 500-600 ml of blood may be rapidly removed via venesection.
6. Control Tachycardia. Give digoxin but watch pulse rate closely. If it is slowed too much the output of the right ventricle may exceed the capacity of the mitral valve.

7. Look for the precipitating cause of the acute failure.

Management of the Cardiac Patient during Labour:

Usually labour is easy. Elective Caesarean section carries great danger in these patients but must be carried out if obstetrical problems exist which will make delivery difficult.

Antibiotics (e.g. Penicillin G O.5 million units b.d.) should be administered throughout labour prophylactically.

First Stage

. Give liberal sedation (e.g. morphine 15 mg) to reduce anxiety.

. Keep the patient propped up and treat any cardiac failure aggesssively. Cardiac failure in the first stage is not in itself a reason for Caesarean section. If, however, delivery is considered to be urgent there are three possible methods:

   1. Use of suction cup.
   2. Incision of cervix under local anaesthesia and extraction of the foetus by forceps or suction cup.
   3. Caesarean section.
      Do not use N_2O/O_2 for analgesic purposes as this will cause reduced oxygen saturation of the blood.

Second Stage

Discourage the patient from bearing down. Carry out an episiotomy to speed delivery and use forceps or the vacuum extractor if delivery has not occurred 20 minutes after full dilatation.

Third Stage

. Avoid oxytocics such as ergometrine. Suture the episiotomy under local anaesthesia.

. Do *not* allow post-partum haemorrhage as a means of reducing blood volume. If necessary, blood may be withdrawn in a controlled manner from a vein. Normally no more than 1 pint should ever be removed in this way. As soon as delivery is complete, prop the patient up and continue oxygen administration.

Puerperium

The majority of maternal deaths due to heart failure in pregnancy occur in the first 24 hours *after* delivery so vigilance should be increased

during this period. During this time, blood which would previously have been filling the chorio-decidual space and supplying the uterus is added to the general circulation and the right ventricle may be overloaded.

Keep the patient hospitalised with bed rest for a minimum of 14 days and do not discharge her until all indications of imminent failure have disappeared. Breast feeding should be avoided.

Valvotomy during pregnancy

Considerable success has been reported in this field. In suitable cases the operation may be performed in the first half of pregnancy.

Prognosis:

Maternal

Cardiac disease accounts for about 3-5% of maternal mortality. If atrial fibrillation occurs 70% will go into failure and the mortality rate rises to 40%. After the age of 35 the risk is doubled in all grades. Patients in grade 1 and 2 failure should be advised to allow 2 years to elapse before contemplating another pregnancy. Patients in grade 3 and 4 should consider sterilisation as further pregnancies are contra-indicated.

Foetal

The prognosis is related to the degree of maternal cardiac failure. The still birth rate is 3%, the neonatal death rate 2% and the incidence of premature labour 43% (compared to 7% over all).

CHAPTER 12

ANAESTHESIA IN OBSTETRICS AND GYNAECOLOGY

General Instructional Objective

Comprehends the use of analgesia and anaesthesia in obstetric patients to enable him to choose the most appropriate technique for each of his patients during labour and delivery.

Specific Behaviours

1. Describes the pain felt by a labouring woman and indicates the physical and psychological factors that may influence it.
2. Knows the clinical pharmacology of the sedative and analgesic drugs given by injection to women in labour and appreciates the limitations and hazards associated with their use.
3. Knows the basic clinical pharmacology of nitrous oxide, trichlorethylene and methoxyflurane as used for intermittent inhalational analgesia.
4. Demonstrates ability to employ Midogas nitrous oxide apparatus, and Recota and Cardiff vapourisers for inhalational analgesia during labour and normal delivery.
5. Knows the anatomy of the pudendal nerve and describes the techniques of pudendal nerve block.
6. Knows the basic pharmacology of Lignocaine.
7. Describes the sensory nerve supply to the uterus and birth canal.
8. Describes the techniques of lumbar and sacral epidural blocks, and exhibits knowledge of dangers and limitations of these procedures.
9. Demonstrates ability to perform local infiltrations of perineum with local anaesthetic agent.

10. Describes the indications for and hazards of general anaesthesia in obstetrics.
11. Demonstrates ability to assist anaesthetist at induction of general anaesthetic in obstetric patients.

General Instructional Objectives

Understands the principles of cardio-respiratory resuscitation to a degree that will enable him to carry out resuscitative procedures effectively.

Specific Behaviours

1. Knows causes of cardiac arrest.
2. Demonstrates ability to perform closed chest cardiac compression and expired air ventilation on training mannkini.
3. Can diagnose and treat acute respiratory insufficiency.
4. Demonstrates knowledge of correct treatment of acute hypovolaemia.

Anaesthesia in Obstetrics and Gynaecology

The Pain of Labour

1. **Physical Factors**

   a. *Dilatation of the Cervix–*

   This is the main cause of pain in the first stage of labour and is due to the resistence of the cervix to the dilating forces. With the first few powerful contractions the discomfort is felt only during the terminal part of the pressure rise in the uterus, and as soon as the uterine muscles start to relax, the pain recedes. However, once painful contractions become established, the discomfort commences as soon as the uterine pressure rises above 20 mm Hg and does not disappear until the intrauterine pressure again falls to 20 mm Hg. (Fig. 12.1) In an average normal labour contractions become painful

when the cervix is 3-4 cms dilated, and as cervical dilatation proceeds, the intensity of the pain increases. It is usually very severe during the last quarter of the first stage. Usually the discomfort of the uterine contractions commence in the back and then spread to the front of the abdomen.

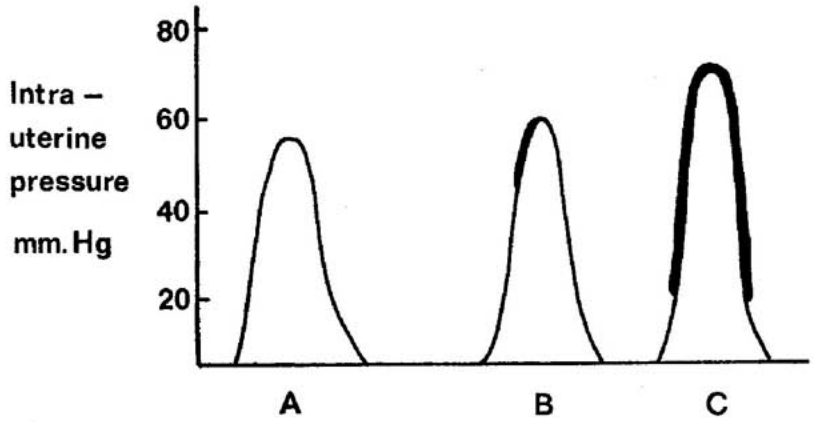


Fig. 12.1. Occurrance of pain in relation to uterine contractions during labour.
A. Painful contractions during late pregnancy.
B. Initial painful contractions.
C. Contractions during established labour.
Duration of pain shown by thickened line.

   b. Contraction and distension of the uterus and perhaps contraction of the circular muscle fibres in the cervix.

   c. Tension in the supporting ligaments of the uterus. Traction on adnexae and the parietal peritoneum.

   d. Reflex skeletal muscle spasm.

   e. Distension of the vagina, vulva and perineum. The beginning of the second stage is usually associated with slightly less pain, but the most severe pain is associated with the delivery of the head.

Situations predisposing to more pain than normal:

a. Elderly primaparity.
b. Presence of dystocia – due to a small pelvis or large baby.
c. Prolonged labour from any cause may result in a lowering of the pain threshold and more severe pain.

d. Fatigue, anaemia, general debility and malnutrition lower the pain threshold.

2. **Psychological Factors**

a. Cultural patterns and customs.
b. Emotional preparedness for labour.
c. Fear, apprehension, ignorance and loneliness.
d. Attitude of the patient's attendants.

3. **Nerve Pathways:**

a. In the first stage of labour the sensory pathways from the uterus and the cervix are the fine unmyelinated fibres accompanying the sympathetic nerves which pass through the cervical ganglia, uterine plexuses, inferior hypogastric plexuses, hypogastric nerves, superior hypogastric plexuses and para-aortic plexuses. From here they pass to the lumbar and lower thoracic sympathetic chain and then via white rami communicantes to the dorsal roots of the 11th and 12th thoracic nerves, and then to the spinal cord.

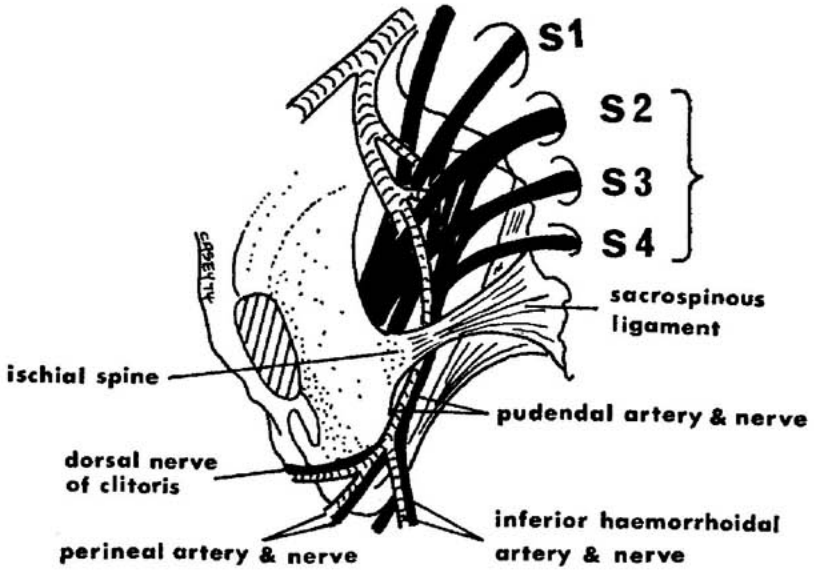


Fig. 12.2. The Formation of the Pudendal nerve from the 2nd, 3rd and 4th Sacral nerves.

b. The pain of the second stage is caused by distension of the birth canal and is conveyed by sensory fibres of the pudendal nerves, which enter the spinal cord via the posterior roots of the 2nd, 3rd and 4th sacral nerves (Fig. 12.2)

4. **Anatomy of Pudendal Nerve**

The pudendal nerve is formed by branches of the 2nd, 3rd and 4th sacral nerves in the sacral plexus. It lies behind the ischial spine on each side medial to the pudendal artery, where it begins to divide into several branches, as shown in the illustration (Fig. 12.3) As it curves forward to the medial aspect of the ischial tuberosity, it gives off the inferior haemorrhoidal nerves, perineal nerves, the dorsal nerve of the clitoris, and the nerve to the labia majora.

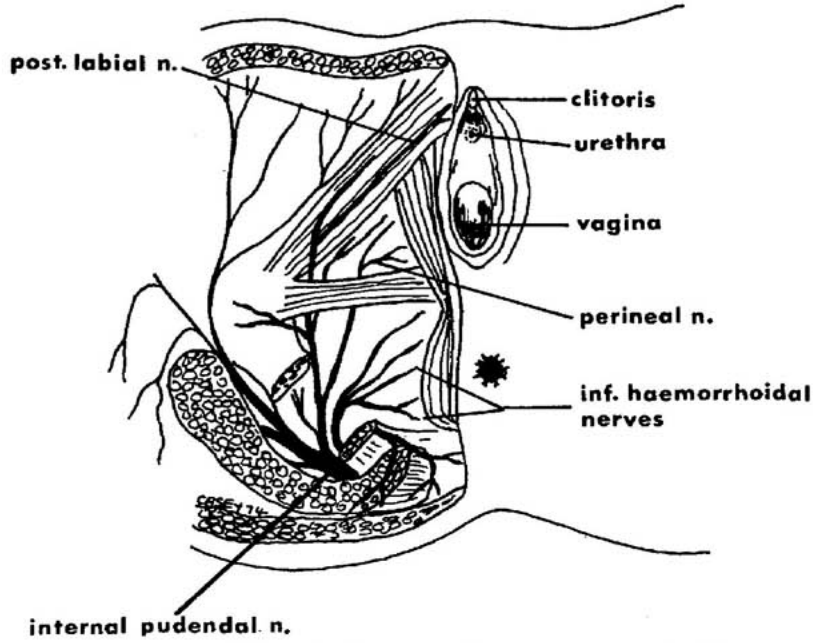


Fig. 12.3. Course of the Pudendal nerve and its branches over the Perineum.

Analgesia

This is aimed at raising the patient's pain threshold (the level of awareness at which any sensation becomes painful) to a point where pain is either eliminated, or at least greatly reduced in intensity.

1. Mild Sedatives and Tranquilizers

a. *Valium* (Diazapam)

Mode of Action	– CNS action reduces anxiety and apprehension. – In higher doses (over 20 mg I.M.I.) it produces skeletal muscle relaxation and may thus reduce skeletal muscle spasm.
Dose	– 20 mg I.M. or orally.
Onset of Action	– I.M. 5-15 minutes. Orally 15-30 minutes.
Duration of Action	– Variable – usually several hours.
Advantages	– Safe, few side effects.
Disadvantages	– Inadequate as an analgesic.

b. *Promethazine HCl*

Mode of Action	– This is an antihistamine with a tranquilizing effect on the CNS. – Anti-emetic.
Dose	– 30-50 mg I.M.I.
Onset of Action	– 5-15 minutes.
Duration of Action	– Variable.
Advantages	– The anti-emetic effects are useful in view of the emetic effects of narcotics and epidural anasthesia.
Disadvantages	– Poor analgesic.

c. *Promazine HCl*

Similar in mode of action and use to Phenergan.

d. *Scopolamine*

Mode of Action	– Acts on the CNS producing sedation and twilight sleep. It may also cause amnesia. – Atropinic effects.
Dose	– 0.4 mg I.M.I.
Onset of Action	– 5-15 minutes.
Duration of Action	– Variable.

2. Narcotic Analgesics

a. *Pethidine HCl*

Mode of Action	– CNS depressant
Dose	– 50-100 mgm I.M.I.
Onset of Action	– 5-15 minutes.
Duration of Action	– 2-4 hours.
Side effects	– Dizziness, nausea, vomiting, rarely hypotensive collapse.
Disadvantages	– Poor sedative action. – Respiratory depression of the infant.

Pethidine may be combined with a tranquillising agent to improve sedation, e.g. Scopolamine 0.4 mgms, or Sparine 25-50 mg. It may also be combined with levallorphan, a narcotic antagonist, to reduce the respiratory depression of pethidine e.g. Pethilorfan.

b. *Morphine Sulphate*

Mode of Action	– CNS depressant
Dose	– 10-15 mgm I.M.I.
Onset of Action	– 10-15 minutes.
Duration of Action	– 3-6 hours.
Side Effects	– Nausea.
Disadvantages	– Respiratory depression of the infant.

c. *Omnopon* (mixture of the anhydrous components of morphine)

Mode of Action	– Similar to morphine
Dose	– 15-20 mgm I.M.I.
Onset of Action) Duration of Action) Side effects) Disadvantages)	– Similar to morphine.

N.B. Narcotic Antagonists

These are used to prevent or reverse respiratory depression of the infant. To achieve this they must be given either I.V.I. to the mother at least 20 minutes before delivery, or to the infant via the umbilical vein or I.M.I.

Lethidrone (Nalorphine)

Dose – 5 mgm to the mother, or 0.5 mgm to the infant.

3. Inhalation Analgesia

This is administered in conjunction with injected analgesic drugs, the mask being held by the patient who inhales intermittently to alleviate the peaks of pain. Inhalational analgesia may be necessary during the last quarter of the first stage and during the second stage, especially at the time of delivery.

It takes 15-30 seconds for these agents to build up in the brain so inhalation should be started at the *onset* of a contraction, rather than when the pain becomes extreme.

Drugs Used

a. *Nitrous Oxide*

This gas is contained under pressure in cylinders its release from the Midogas apparatus being activated by the patient inhaling from a closely fitting mask. She should be permitted to hold the mask herself, so that if she becomes unconscious it will fall away from her face.

Action	– CNS action producing analgesia – then anaesthesia, if a high concentration is inhaled continuously.
Dose	– The Midogas apparatus allows a maximum concentration of 70% N_2O with 30% O_2. At this concentration, provided that maternal respiration and circulation are adequate, maternal or foetal anoxia should not occur.
Onset of Action	– May take up to 30 seconds, especially if administering less than 70% concentration.
Duration of Action	– Diminishes almost completely within 60 seconds of cessation of inhalation.
Advantages	– Quick action. – Safe with adequate O_2. – No interference with uterine action. – No foetal depression. – Does not build up in the body – no time limit to use.

	– Suitable for cases with cardiac or pulmonary pathology.
Disadvantages	– High variation of individual susceptibility. – Expensive. – Complicated equipment which must be checked often.

b. *Trichlorethylene* (Trilene)
This is a colourless liquid to which a blue dye has been added for identification.

It is vapourized in air by means of a special vapourizer which maintains a constant concentration of 0.5% Trilene: i.e. the Tacota vapourizer.

Action	– CNS effect producing analgesia. If the concentration is high enough or continuous inhalation carried out anaesthesia may result.
Dose	– 0.5% concentration in air, inhaled intermittently.
Onset of Action	– Up to 30 seconds.
Duration of Action	– Significant blood concentrations are present for 20 minutes to 2 hours after discontinuation of inhalation.
Advantages	– Inexpensive – Non-inflammable. – More effective than N_2O. – Relatively simple apparatus.
Side effects and Disadvantages	– Cumulative effect – prolonged use makes the mother and infant drowsy and it should therefore not be used for more than 4 hours unless in reduced concentration. It must *not* be used with a Soda Lime absorption cannister since a neurotoxic substance, dichloracetylene, may be formed, leading to cranial nerve palsy. Consequently, if a general anaesthetic is to be administered, it must be done

with the absorption cannitser excluded, which imposes a serious obstacle.

c. *Methoxyflurane*

Action	– Similar to N_2O and Trilene.
Advantages	– May be used in conjunction with a Soda Lime absorber.
Disadvantages	– Pungent odour.

Points to Remember in Administering Inhalation Analgesia:

a. Antenatal education in use of apparatus, with revision when presented to labour ward, is advisable.

b. Check:

i. Gas and O_2 taps turned on,
ii. Machine is switched on,
iii. For Trilene check the contents of the vapourizer.

c. Check the fit of the mask on the face. If not held on firmly, air will leak in and reduce the effectiveness of the gas.

d. Ensure that inhalation starts as soon as the contraction is felt.

e. Check the expiratory valve (it may stick).

4. **Local Anaesthesia**

Definition:

Local anaesthesia is produced when a drug or drugs react to produce a reversible blockade of conduction in nerves. Conduction in all types of axons is blocked, but in general, small diameter fibres are more susceptible to their action and are slower to recover than those of larger diameter.

Lignocaine

Action	– Probably inhibits the transient increase in permeability of excitable membranes to Sodium Ions.
Metabolism	– Hydrolized by pseudocholinsterase it then undergoes biotransformation in the liver to be excreted partly in bile and partly in urine.
Dose	– Maximum safe dose is 200 mgm (3 mgm / kg b.w.) this being raised to 500 mgm /kg b.w.) if the Lignocaine is combined with 1/250,000 Adrenaline. Thus if 0.5% (0.5 gm in 100 mls) lignocaine with 1/250,000 Adrenaline is used the maximum safe dose is 100 mls. Lignocaine is available in concentrations of 0.5%, 1% and 2%.
Onset of Action	– Almost instantaneous.
Duration of Action	– About 30-60 minutes with local infiltration and regional block, and about 60 minutes with epidural block spinal anaesthetic.
Advantages	– Potent local anaesthetic. – Often produces sedation along with the analgesia.
Disadvantages	– Local nerve injury– i. due to contaminants of the lignocaine preparation or as a result of adrenaline ischaemia. ii. caused by technique of intraneural injection or direct needle trauma. – Toxic reaction – caused by a high plasma level and its effects on the CNS and CVS. A mild reaction is shown by pallor, anxiety, nausea and restlessness, which may be difficult to distinguish from simple nervousness.

Severe Systemic Reaction:

C.N.S. – Stimulation leading to convulsions, followed, by drowsiness, unconsciousness, medullary depression, apnoea, and vasomotor collapse.

Cardiac – Direct myocardial depression leading to hypotension and cardiac arrest.

Often the first observed indication of any overdosage is a sudden convulsion.

The most severe reaction is cardiorespiratory arrest. Allergy or sensitivity to local anaesthetics are very rare reactions nearly always being caused by overdosage.

Prevention of Systemic Reaction:

i. Use the minimum effective concentration and the smallest volume. Do not exceed maximum safe dosage. CHECK BEFORE INJECTING.

ii. Use vasoconstrictors. These prolong the action and result in lower plasma levels.

iii. Avoid intravascular injection.

Treatment of a Systemic Reaction:

i. oxygen 100%.

ii. Succinyl choline 25-50 mgm if major convulsions occur.

iii. Vasopressor to maintain BP, e.g. Metaraminol 2 mgm I.V. if necessary.

iv. Closed chest cardiac massage if necessary.

Use of Local Analgesia in Obstetrics and Gynaecology:

Indications for use–

. Maternal distress and pain in dystocia.
. Instrumental delivery.

Method of Using Local Analgesia:

a. *Local Infiltration of Perineum*

Its advantages are summarized as follows–
. Practically no anaesthetic mortality.
. Foetal mortality or hypoxia not caused.
. Simplicity of administration.
. Uterine contractions not impaired.
. No interference with the desire to "bear down" during labour.
. Toxic effects are minimal.

In the case of spontaneous delivery the patient is placed in the lithotomy position. The fourchette and the adjoining area are first infiltrated using a 5 cm 20 guage needle. The injections are then made in a fan shaped manner as illustrated in Fig. 12.4.

Alternatively, if analgesia is required simply to perform an

episiotomy, only the area to be incised need be infiltrated, 0.5% lignocaine is used, combined with 1/25,000 adrenaline; the maximum amount being 100 mls although 50 mls should suffice.

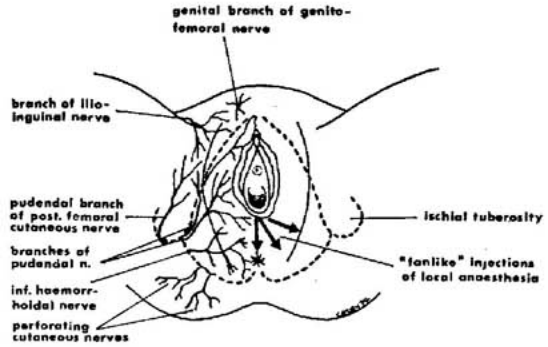


Fig. 12.4. Innervation of the perineum and illustration of "fanlike" infiltration of perineum with local anaesthetic.

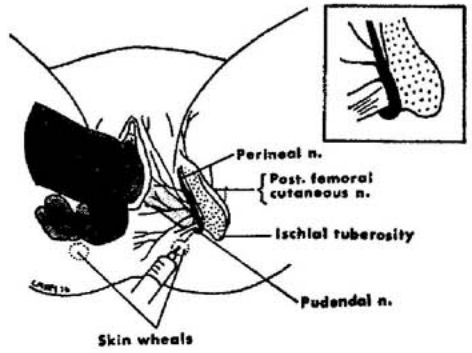


Fig. 12.5. Transperineal Pudendal Block.

b. *Pudendal Block*

i. *Transperineal–*

First a small amount of subcutaneous tissue is infiltrated on each side midway between the anus and the ischial tuberosity. The index finger of the left hand is inserted into the rectum (or vagina) and the left ischial spine is palpated. A 20 guage needle, 10 cms long, is guided to

a point just below and beyond the spine. After drawing back, to exclude the possibility of intravascular location 15 ml of 1% lignocaine is injected to anaesthetise the pudendal nerve (Fig. 12.5).

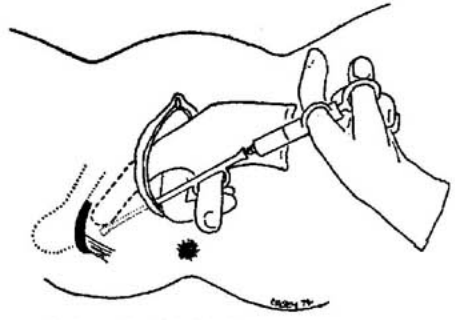


Fig. 12.6. Transvaginal Pudendal block.

ii. *Transvaginal–*

A transvaginal guarded needle is guided to the ischial spine as shown in the diagram. It is held with the point just posterior and medial to the tip of the spine. The needle is then advanced until it touches the sacro-spinal ligament which is infiltrated with 2 or 3 mls of 1% lignocaine. It is then advanced through the ligament and about 15 mls of 1% lignocaine is injected (Fig. 12.6).

Advantages of Pudendal Block

. Relatively simple and painless procedure. Anaesthetises large area of vagina and perineum.

Disadvantages of Pudendal Block

. Considerable failure in anaesthetizing the pudendal nerve.
. It may eliminate the desire to "bear down" during the second stage of labour.

iii. *Epidural Anaesthesia*

Method – The patient lies on her side and is aided by an attendant to flex her spine. An area of her back, including and surrounding the lower lumbar vertebrae, is swabbed and draped by a gowned and gloved operator.

A short-bevelled 22-26 guage needle is then slowly advanced through the 4th or 5th lumbar interspace. The resistance of the ligamentum flavum will be felt, during which time air cannot be injected with ease. However, when this ligament is penetrated and the epidural space entered, air is injected easily, (this is called the air rebound technique), whereupon 25-50 mls of 1% lignocaine in 1/250,000 adrenaline is injected. The amount is calculated according to the patient's height so as to anaesthetize the spinal roots from the level of the 10th thoracic to the 5th lumbar vertebrae.

Alternatively, epidural anaesthesia may be administered via the caudal or sacral route.

Duration of Action – 1-12 hours depending on whether a "single shot" or "continuous catheter" is used.

Advantages
- Pain in all stages of labour is completely eliminated.
- May be conveniently topped-up if the needle is taped in position.
- Patient remains fully alert.

Dis-advantages
- Maternal hypotension may result in foetal hypoxia if the systolic B.P. decreases to below 90 mm Hg. (this occurs in about 5% of cases). The incidence of maternal hypotension may be reduced by having the patient lie on her side for as much of the time as possible. This prevents inferior vena-caval obstruction.
- Patient cannot tell if the contractions are excessively prolonged or intense. Danger of subarachnoid anaesthesia and possible precipitous drop in B.P. Thus a patient with epidural spinal anaesthesia must have her B.P. and uterine contractions constantly monitored, the latter being *continuously* monitored if syntocinon is also being used.
- Danger of uterine rupture if obstruction to labour is present with intense uterine

contractions. The patient cannot communicate if there are any symptoms of impending rupture.

5. General Anaesthesia

Indications
- Caesarean section.
- Need for full uterine relaxation – full breech extraction or to facilitate the delivery of the after-coming head in a breech delivery; correction of transverse lie.
- Manual extraction of the placenta.
- When it is desired for aesthetic reasons that the patient should remain unconscious (e.g. delivery of deformed infant).

Advantages
- Fast.
- Reliable.

Disadvantages
- Foetal depression.
  i. directly due to anaethestic agents passing the placental barrier.
  ii. secondary to maternal hypoxia if this is present.
- Danger of aspiration – labour ward patients often have a full stomach. This must be assumed in all labour ward patients. Although a stomach tube may be passed to remove stomach contents, it must never be relied upon. Cricoid pressure should be maintained by an assistant from the time of induction until an endotracheal tube has been inserted and the cuff inflated.

  As a prophylactic measure no solid foods or hypertonic fluids should be administered during labour. If aspiration does occur one or both of the following two problems may result:

  i. Immediate obstruction. Treat with suction, posture (head dependant) O_2 administration under pressure. Follow up with bronchoscopy.
  ii. Mendelson's syndrome. After the inhalation of a small volume of gastric contents there is a latent period of about an hour followed by acute inhalation pneumonitis. This is characterized by cyanosis, tachycardia, dyspnoea, and pulmonary oedema. Treat with O_2 administration and 200 mgm of of hydrocortisone I.M.I.

- Patient may be dehydrated and ketotic due to the effect of a prolonged labour with inadequate fluid and sugar intake. This, combined with the stress and blood loss of surgery predisposes to shock and serious complications such as acute renal failure and Sheehan's post-partum pituitary necrosis. Dehydration and ketosis should be corrected by administration of I.V. glucose solution.

General Anaesthesia for Caesarean Section

Blood must be cross-matched for all Caesarean sections and if placenta praevia is suspected a drip must be running and blood immediately available. The average blood loss for an uncomplicated Caesarean section is 500-1000 mls.

a. *Premedication*

- Atropine 0.6 mgm or Scopolomine 0.4 mgm.

or
- Haloperidol (SERENACE) 1.5 mgm tab. or injection.

b. *Pre-Oxygenation*

- The patient breathes 100% O_2 while she is being catheterized and the abdomen swabbed and draped.

c. *Induction*

- Thiopentone 200–350 mgm (5 mgm/kg body wt) followed quickly by Succinyl choline 75-100 mgm. Cricoid pressure is applied by an assistant until tracheal intubation is completed.

Drugs used during induction–

Thiopentone (sodium thiopentone)

Action
- Hypnotic action on the CNS.

Dose
- As above, i.e. 5 mgm/kg body weight in 2.5% or 5% solution.

Onset of Action
- Within 30 seconds of intravenous injection. There is little or no stage of excitement with this anaesthetic.

Duration of action
Relatively short.

Advantages
- Quick, pleasant induction.
- Rapid recovery.
- A single "sleep dose", although it crosses the placenta readily, appears to have a minimal effect upon the infant.
- Good uterine relaxation.

Disadvantages
- Short acting.
- Not adequate for skeletal muscle relaxation nor for analgesia.

Contra-indications
- Hypersensitivity to barbiturates.
- Severe asthma or other respiratory disease.
- Porphyria.

N.B. Because thiopentone sensitivity or overdose may result in severe respiratory depression, it must only be administered if resuscitative equipment is available.

Succinyl choline

Action – Selective depolarization of the neuro-muscular junction.

Dose – As above, i.e. 75-100 mgm I.V.

Onset of action – 30-60 seconds.

Duration of action – 5-10 minutes.

Advantages
- Complete and rapid muscle relaxation which facilitates intubation.
- Muscle relaxants do not cross the placenta and thus do not affect the foetus.

d. *Maintenance*

- Nitrous Oxide and Oxygen (5 litres: 2 litres) with moderate hyperventilation.
  Tubocurarine (20 mgm) or Alcuronium (10 mgm) may be given as the effect of succinyl choline is wearing off.

After the delivery of the patient anaesthesia may be deepened by the addition of cyclopropane or intravenous pethidine. Neostigmine (an anticholinesterase) will be necessary finally as an antidote to the muscle relaxants.

N.B. Halothane is avoided in Caesarean section because uterine relaxation is not required and will predispose to excessive post-partum haemorrhage.

Drug Used during Maintenance

Cyclopropane

Action – Hypnotic effect on the CNS.

Dose – 10% concentration is adequate for maintenance.

Duration of action – Return to clouded consciousness occurs after 5-15 minutes.

Advantages – Does not produce uterine relaxation.

Disadvantages
- i. Explosive (cannot be used with diathermy)
- ii. May cause foetal depression.
- iii. Nausea and vomiting during recovery.
- iv. May lead to cardiac arythmia.

General Anaesthesia for Forceps Delivery:

Usually epidural or other forms of local anaesthesia will be used but general anaesthesia may be desirable, especially where speed or uterine relaxation are required.

Requirements
- Maximal oxygenation.
- Minimal narcosis.
- Relaxation of the pelvic floor.
- Uterine relaxation if requested (uncommon).

Technique – Mother breathes 100% O_2 via face mask from a Boyle's machine. Check suction, laryngoscope, all connections.

Insert Mitchell, or similar needle, into hand or arm vein. Inject Atropine 0.6 mgm. I.V., followed by Thiopentone 5 mgm/kg b.w., and Succinyl choline 75-100 mgm. Assistant applies cricoid pressure until the endotracheal tube is inserted and the cuff inflated. The patient is ventilated with N_2O (70% with 30% O_2).

Paralysis is then maintained by intermittent injections of Succinyl choline in doses of 20 mgm. Once

delivery is completed, spontaneous respiration is allowed to return and anaesthesia may be deepened with cyclo-propane during the repair of the episiotomy. The endotracheal tube should not be removed until the patient is on her side and showing signs of lighter consciousness.

Ergometrine, unless contra-indicated by pre-eclampsia, high B.P. or by the administration of a vasopressor agent, is administered after the delivery (0.5 mgm I.M.I.).

General Anaesthesia when Uterine Relaxation is Required

a. For breech extraction or assisted breech delivery:

- A Mitchell, or similar needle, is inserted into a vein and when the obstetrician is ready to apply the forceps, 150-300 mgm of Thiopentone is injected rapidly. Usually this is all that will be required.

An anaesthetic machine, intubation equipment and suction should always be prepared ready for use, if necessary.

b. For manual removal of the retained placenta:

- If severe blood loss has been experienced but has abated the loss should be replaced before the anaesthetic is begun. Proceed as for the forceps delivery. Ether or Halothane may be required to relax the uterus if oxytocics have been given.
If heavy bleeding is continuing ergometrine 0.5 mgm I.M.I. should be given, followed by anaesthesia and removal of the placenta. Ideally the ergometrine will have started its effect soon after the procedure is completed.

Drug Used –

Halothane

Action – C.N.S. hypnotic.
Dose – 2% concentration in air.
Advantages
- Good skeletal muscle relaxant and uterine relaxant.
- No apparent tendency to cause nausea and vomiting.

Disadvantages
- Cardiorespiratory depression.
- Uterine relaxation may predispose to post-partum haemorrhage.
- Followed by hepatic failure in rare cases.

CARDIAC ARREST AND HYPOVOLAEMIA

Cardiac Arrest

Definition:

Failure of the heart to maintain an adequate cerebral circulation in any situation other than that caused by progressive and irreversible disease.

Causes:

1. **C.N.S.**
   a. Effect of analgesic or anaesthetic agents on cardiac and respiratory centres of the medulla.
   b. Cerebrovascular accident, e.g. in eclampsia.
2. **C.V.S.**
   a. Depressant effect of drugs on myocardium.
   b. Heart failure due to increased peripheral resistance and/or reduced venous return.
   c. Acute cor pulmonale following pulmonary oedema.
   d. Myocardial infarction.
   e. Acute hypovolemia due to blood loss or endotoxic shock. Severe shock with cardiac arrest is more likely if there is pre-existing anaemia.
3. **Respiratory Failure**

   This is usually secondary to pulmonary oedema or drepession of the mudullary respiratory centre.
4. **Electrolytic Disturbance**

   Especially hypo- or hyperkalaemia.

Management:

1. **Make the Diagnosis**

   Absent carotid pulse, unconsciousness and widely dilated pupils within 6-8 seconds, cessation of respiration within 10-20 seconds. At this stage cerebral circulation is inadequate and irreversible brain damage will occur if arrest continues for 3-4 minutes.

2. **Ventilation**

   a. Place patient on her back on a firm surface – the floor if necessary

   b. Clear the airway – head to one side, remove false teeth, food, mucus, etc.

   c. Mouth to mouth breathing – with head tilted back, nose pinched closed and jaw held forwards. The chest should rise visibly. Repeat rapidly at first but then continue at about 15 breaths per minute.

   d. Air-Viva bags are available in each ward. If one is handy it may be used in place of mouth to mouth breathing. When using it ensure –

      . Firm fit of face mask,
      . Clear air-way (head tilted well back, jaw held forward).
      . Good chest expansion with each bag inflation.
      . Abrupt bag release for exhalation.

      Set the oxygen flow at 10 litres/minute.

   e. Endotracheal Intubation – when a skilled operator arrives.

3. **External Cardiac Compression**

   a. Elevate legs 30° to improve venous return. Patient must still be on a hard surface.

   b. Using the heel of one hand, reinforced with the other hand on top of it, press the lower third of the sternum sharply back for 1-2 inches and then release rapidly. Repeat this vigorously 60 times per minute pausing only to allow ventilation. (If alone, 2 inflations to 15 chest compressions). An assistant should check that a femoral pulse is palpable. The amount of circulation produced by this manouvre is 20-40% of normal.

4. **Intravenous Sodium Bicarbonate Drip**

   Give 100-200 mg stat. in a concentrated solution.
   Calculate the subsequent dosage by:

$$\text{No. of mg} = \frac{\text{wt (kgm)}}{10} \times \text{Duration of arrest (minutes)}$$

5. Attach ECG monitor and set up defibrillator and begin drug therapy:

   a. *In case of Ventricular Fibrillation*

      Apply generous amounts of paste to the electrodes, massage paste over the skin, place one electrode just below the right clavicle and the second just over the apex of the heart. Apply firm pressure. Ask everybody to avoid contact with the patient and the bed. Charge the defibrillator to 200 joules or more then discharge the countershock by the switch.
      If unsuccessful give 5 mls of adrenaline 1/10,000 I.V. and repeat shock.

   b. *In case of Ventricular Asystole*

      Give calcium chloride 5 mls of 5% I.V. and adrenaline, 5 mls of 1/10,000 I.V. Both drugs may be repeated in 5-10 minutes.

   c. *In case of electrolytic disturbance*

      i. Low Potassium – give potassium 10 mgm. slowly I.V. and propanolol 1-5 mgm slowly with Atropine 1-2 mgm.

      ii. High Potassium – give calcium chloride 5 mls of 10% I.V.; and give insulin with glucose (3 gm glucose for each unit of insulin).

6. **Transfer patient to Intensive Care Unit**

   Give appropriate care to prevent recurrence and treat sequelae:

   a. Cerebral oedema) Give 20% Mannitol 50-200 mls.
   b. Renal damage (acute tubular necrosis)) over 4 hours. Also give hydrocortisone or betamethasone in massive doses. Hypothermia to 32° has limited use.
   c. Trauma to ribs and sternum and perhaps liver.

Management of Acute Hypovolaemia:

1. Resuscitate as necessary; begin an ECG monitor.
2. Replace lost fluids, using blood if possible (SPPS until blood becomes available). Do not overtransfuse (check CVP).

3. Monitor urinary output. If it is below 15 mls per hour then oliguria exists, and acute renal failure is imminent. If renal shutdown not advanced, fluid replacement should lead to the return of a normal urine output (at least 60 mls/hr).
4. If urinary output does not rapidly return to normal, or if renal failure is anticipated use a diuretic such as frusemide. The dose is 20-400 mg I.V.I. Diuresis can be expected within an hour and the drug continues acting for about 2 hours.
5. Serum electrolyte levels should be monitored closely with special attention being paid to the serum potassium levels. Significant variations in this electrolyte may cause cardiac arrhythmias or cardiac arrest.

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CHAPTER 13

LEUCORRHOEA AND PELVIC INFECTION

General Instructional Objective

Applies bacteriological knowledge and gynaecological skills to indicate the management of patients with leucorrhoea or infection of the female genital tract.

Specific Behaviours

1. Describes the types of infection which might affect the female genital tract.
2. Explains the differences between physiological and pathological vaginal discharge.
3. Diagnoses vaginal discharge and pelvic infections.
4. Suggests appropriate investigations related to the vaginal discharge or pelvic infection.
5. Suggests appropriate therapy for the infection.
6. Explains social implications of the disease process.

Leucorrhea and Pelvic Infection

Vaginal Discharge

1. **Physiological Discharge**

   The vagina in a woman of reproductive age normally contains secretions consisting of:

a. Vaginal transudate.
b. Cervical mucus.
c. Uterine secretion.
d. Secretion from Bartholin's glands.
e. Fallopian tube secretion.

At puberty oestrogen stimulus causes the secretion of glycogen from vaginal squamous epithelium. In this vaginal transudate the Döderlein's bacillus (lacto-bacillus) become active and convert the glycogen to lactic acid resulting in a pH of 4.0 to 5.5. Under the influence of pregnancy or oral contraceptives glycogen production increases and the pH falls to between 3.5 and 4.5. Following the menopause the vagina returns to it's pre-pubertal state with absent glycogen and a consequent pH of 6 to 8.

An increased production of these normal secretions is the most common cause of symptomative vaginal discharge and is termed leucorrhoea. The main causes are:

a. Hypersecretion of the cervical glands associated with:
   i. Pelvic congestion – may be due to pelvic pathology or may be psychosomatic.
   ii. Cervical ectopic columnar epithelium – may be related to increased oestrogenic stimulus causing hypertrophy of endocervical glands.
   iii. Oestrogenic oral contraceptives.
   iv. Pregnancy.
   v. Ovulation cascade.
b. Uterine secretion – before and after menstruation.
c. Bartholins glands – activated by sexual excitement.
d. Increased vaginal transudation due to sexual excitement.

2. **Pathological Discharge**

a. *Infective*

i. *Trichomonas Vaginalis vaginitis.*

This infection is present in about 15% of women presenting with a discharge. The discharge is classically *frothy, yellow green* and *offensive*. It may be associated with a *pruritis* so severe that scratch marks may be seen or the discharge might be slightly blood stained. Possible associated findings are dysuria and an acutely inflamed vagina and cervix (strawberry cervix). It may be asymptomatic.

ii. *Monilial Vaginitis* (thrush)

This causes a thick, creamy or yellow-white discharge. The associated irritation causes a desire to rub the vulva rather than scratch it. Husbands may complain of post-coital itching. On examination the vagina is classically inflamed with white, cheesy plaques of discharge which on rubbing away leave multiple haemorrhagic spots.

iii. *Neisseria Gonorrhoea*

This discharge is yellow and purulent. In advanced cases symptoms of salpingitis or pelvic peritonitis may be present. The patient may complain of dysuria. On examination the cervix will usually be reddened and covered with exudate, the urethra will be reddened and Skene's and Bartholin's glands may be thickened and palpable.

iv. *Childhood Vaginitis*

This causes a purulent vaginal discharge in a pre-pubertal child which may be associated with soreness of the vulva, and dysuria.

v. *Atrophic (Senile) Vaginitis*

A yellow or purulent discharge which may be blood stained is complained of by a post-menopausal woman. There may also be vulval or perineal discomfort. On examination the vaginal epithelium is thin and atrophic with multiple reddened areas, mostly seen around the vault.

vi. *Chronic Cervicitis*

This may cause a thick, tenacious, mucopurulent discharge. Other symptoms include backache or low abdominal pain, post-coital bleeding, menorrhagia and congestive dysmenorrhoea. It will be diagnosed because of the presence of an inflamed, tender cervix in association with the discharge. It should not be confused with the over-production of normal mucus by ectopic columnar epithelium.

vii. *Others*

Occasionally puerperal pyometra, or hydrosalpinx will discharge via the vagina.

b. *Traumatic*

i. Due to chemical irritation, e.g. Dettol, household acidic solutions used as spermicides, etc.

ii. Burns – douching with hot fluids.

iii. Abrasions and lacerations – commonly following intercourse or childbirth.

The nature of the discharge in each case will vary depending on the degree of secondary infection but the diagnosis should be evident from the history.

c. *Malignancy*

Malignancy of the vagina, cervix, uterus or fallopian tubes may cause a mucopurulent or haemorrhagic vaginal discharge. Ovarian tumours may cause a bloody discharge secondary to hormonal effects on the endometrium.

d. *Foreign Bodies*

These are most common as a cause of vaginal discharge in children.

e. *Fistulae*

These may be urinary or faecal and typically follow radiation, major gynaecological surgery, diverticulitis, Crohn's disease or, "less commonly" a difficult confinement.

f. *Other Causes*

i. *Granulations*

These may occur in the vaginal vault following total hysterectomy. They cause a thin, mucopurulent discharge.

ii. *Vaginal, cervical and uterine polyps, and submucous fibroids*

These may undergo ulceration and infection causing a foul purulent discharge.

iii. *Abortions*

Following abortion retained products of conception may undergo autolysis and become secondarily infected leading to a haemopurulent discharge.

Infections of The Female Genital Tract

1. **Infections Producing a Discharge**

a. *Trichomonas Vaginalis Vaginitis*

Incidence : Most common vaginal infection. 5%-10% of women are carriers and it is found in 15% of women presenting with a discharge.
Organism : T. vaginalis is a motile flagellate.
Pathogenesis : Usually contracted during coitus but also transmitted by towels, toilet seats, fingers, baths, etc. The optimum pH for growth is 5.5 to 6.5.
Signs and Symptoms : See "Pathologic Discharges".
Diagnosis : Usually possible on clinical findings.
Also: i. Microscopy of wet smear preparation.
ii. Pap smear – Trichomonads seen in 2/3 of infected patients.
iii. Culture in a "trichomonads" medium followed by wet smear preparation.
Treatment : i. Metronidazole (Flagyl) 200 mg t.d.s. orally for 14 days for both husband and wife.
ii. Floraquin pessaries b.d. for one week.

b. *Monilial Vaginitis* (Thrush)

Incidence : Second most common vaginal infection. 10-15% of women are vaginal carriers. It may be combined with trichomonas vaginalis infection.
Organism : Candida albicans, a small Gram-positive fungus. It also prefers an acid medium (pH 5.0 to 6.5) and abundant glycogen.
Pathogenesis : The infection may be transmitted similarly to trichomonas vaginitis, or endogenously, from the patient's own gastro-intestinal tract. Clinical infection probably follows alteration in the vaginal environment due to:

i. Pregnancy – causes increased glycogen and acidity.
ii. Oral contraceptives – also increases glycogen and acidity.
iii. Diabetes mellitus – causes glycosuria.
iv. The use of broad spectrum antibiotics – causing an alteration in the vaginal flora.

Signs and Symptoms : See "Pathologic Discharges".

Diagnosis : Usually clinical. Also:
i. Microscopy – Hyphae may be seen with Gram stain.
ii. Pap. smear – will reveal 50% of infections.

Treatment : i. Alter the cause if possible, e.g. Diabetes mellitus, oral contraceptives.
ii. Mycostatin (Nystatin) pessaries. 1 nocte for 14 days.
iii. Floraquin vag. pess. 1 b.d. for one week.
iv. Other local medications include Mycostatin powder for the vulva; Natamycin (Pimafucin) pessaries; Amphotericin B vaginal tablets and 1% aqueous Gentian Violet.
Mycostatin (Nystatin) oral tablets 1-2 t.d.s. for 21 days to prevent re-infection from G.I.T. Monilial balanitis in the husband may be treated with Natamycin cream or Mycostatin ointment.

c. *Neisseria Gonorrhoea Infection*

See section on "Pelvic Infection" in this chapter.

d. *Childhood Vaginitis*

Incidence : Uncommon

Organism : Any combination of staphylococci, streptococci, E. coli, pneumococcus, Haemophilus vaginalis or even trichomonas vaginalis. N. gonorrhoea causes the most serious infection.

Pathogenesis: Organism from adults, other children or foreign bodies invade the thin vaginal epithelium.

Signs and Symptoms : See "Pathologic Discharges".

Diagnosis : Exclude foreign body and take vaginal swabs for culture and microscopic examination (avoid trauma to hymen).

Treatment : i. Remove any foreign body.
ii. Oral antibiotics according to sensitivities.
iii. Oestrogen (e.g. dienoestrol) 0.3 mg t.d.s. for 10 days.
N.B. Avoid local treatment in children for psychological reasons.

e. *Atrophic (Senile) Vaginitis*

Incidence : Fairly common.

Organism : Similar to those causing childhood vaginitis.

Pathogenesis: The atrophic epithelium has a low resistance to infection.

Signs and Symptoms : See "Pathologic Discharges".

Diagnosis : Pap. smear – to exclude malignancy.
Vaginal swabs for culture and microscopic examination.

Treatment : Local oestrogen treatment may be used. The usual treatment is to administer oestrogen tablets in a sufficient dosage to improve the vaginal epithelium without including endometrial hyperplasia or bleeding.
Stilboestrol 0.5 mgm daily for 25 days in each 30 or Ethinyl oestradiol 0.01 mgm daily for 21 days in 28 or Premarin 0.625 mgm for 25 days in each 30 may be given.

f. *Chronic Cervicitis*

Incidence : Causes symptoms in about 2% of parous women.

Organism : Most commonly stretpococci, staphylococci and E. coli.

Pathogenesis: It may be due to infection of ectopic columnar epithelium (which has a low re-

sistence to infection) or may occur following trauma due to pessaries, other vaginal appliances, or labour. Many cases diagnosed as having chronic cervicitis really only have increased production of normal mucus from ectopic columnar epithelium.

Signs and Symptoms : See "Pathologic Discharges".

Treatment

i. Cervical cauterisation – this is by diathermy under anaesthesia. Possible complications of this treatment (and cold knife surgery) are secondary haemorrhage and, at a later stage, cervical stenosis.

ii. Trachelorrhaphy – only used when the cervix is badly lacerated.

N.B. Antibiotic treatment is usually not effective.

2. Venereal Diseases

a. *Gonorrhoea* – see "Pelvic Infection".

b. *Syphilis.*

Incidence : Treponema Pallidum is an anaerobic spirochaete requiring moisture and tissue for survival.

Pathogenesis : Syphilis is strictly a venereal or congenital disease. Transmission is by blood or exudate into the vagina via minute lacerations or during menstruation, and into the foetal circulation leading to congenital syphilis. Physiological secretions are not infectious. The incubation period averages 3 weeks duration (1-10). The primary stage (chancre) lasts 1-5 weeks and may overlap with the secondary stage which lasts about 2-6 weeks. This is followed by a quiescent phase lasting 20 years or more, which may be followed by the later or tertiary stage.

Signs and Symptoms :

i. Primary syphilis. The typical chancre is a single small erosion or deep ulcer occurring anywhere from the vulva to the cervix. It is usually accompanied by painless non-suppurative rubbery satellite lymphadonopathy. The lesion on the vulva, unlike syphilitic chancres elsewhere, may be painful.

ii. Secondary syphilis. This is the most highly contagious stage. The eruption assumes many forms but is almost never vesicular. The areas usually involved are the oral cavity, palms and soles and the genital area where the lesion may become vegetative (condylomatous). Accompanying generalised adenopathy is an important sign.

iii. Tertiary syphilis. This may involve any part of the body but most significantly affected are the cardiovascular and central nervous systems.

iv. Congenital syphilis. Untreated acquired syphilis in the mother causes a congenital infection in the foetus but this never occurs before 16 weeks gestation. Adequate treatment of the syphilis before 16 weeks will prevent congenital infection. Treatment after 18 weeks will eliminate the spirochaete from the foetus but cannot prevent possible osseous stigmata.
If the syphilis remains untreated then 25% will die (usually occurs late in pregnancy), 25-30% of live born neonates will die shortly after birth, and about 40% will develop late symptomatic syphilis.
The congenital syphilitic child born alive of an untreated mother usually appears normal until the second to sixth postpartum week.

Investigations :

i. Every pregnant woman must have serological tests for syphilis.

ii. For patients presenting with primary syphilis:

. Darkground examination of the exudate from the sore.

. Test for Wasserman antibody. This does not appear in the blood until the sore is 1 week old.

iii. For the patient presenting with secondary syphilis all the serological tests will be reactive. The highest titres occur about 3 months after infection.

Serology : Three distinct antibodies may be tested for, although they may not all be present in the one serum at the one time.

i. Wasserman antibody (anti-lipid).
Detected by:
W.R.
V.D.R.L.
Kahn & Kline tests

ii. Anti-Treponema antibody.
Detected by:
Reiter Protein Complement Fixation Test

iii. Immobilizin.
Detected by:
Treponema Immobilisation Test

Treatment : Any of the following will achieve clinical cure in 92-94% of cases.

i. Benzathine Penicillin (Bicillin)
2-4 million units I.M. at one session into 2-4 sites.

ii. Aqueous Procaine Penicillin G. 600,000 units daily for 8 days.

Alternatively, the amino glycosides may be substituted.

To check the efficiency of treatment the following tests are essential.

i. Before treatment: – Darkground examination of an accessible lesion.
– Serological tests for each of the 3 antibodies with at least one quantitative test.

ii. After treatment: – Quantitative serological tests monthly for 6-9 months and every 3 months thereafter for at least 2 years.

iii. Follow up: – Should include spinal fluid examination.

c. *Lymphogranuloma inguinale*

Incidence : Occurs in tropical countries – especially in Negroes.

Organism : Virus.

Symptoms and Signs : After an incubation period of 3-21 days a painless papule or ulcer forms on the vulva. This disappears as the inguinal glands enlarge then break down forming sinuses and abscesses with surrounding induration and lympahtic obstruction. Sequelae include elephantiasis, vesico-vaginal fistulae and rectal fistulae. This process may be confused with malignant disease.

Diagnosis: From history, presence of eosinophilia and by elicitation of a positive intradermal Frei test.

Treatment : Chloramphenicol, Sulphonamide or tetracyclines for 2 weeks.

d. *Granuloma Venereum*

Incidence : As for lymphogranuloma inguinale.

Organism : Virus ("Donovan bodies")

Signs and Symptoms : After an incubation period of 7-60 days a chronic granulating lesion develops on the vulva, labia and peri-anal skin. It heals slowly with fibrosis, scarring and fenestration of the nypmhae. The inguinal glands may enlarge but do not suppurate.

Diagnosis : From the history and the finding of Donovan bodies in pus cells from the discharge.

Treatment : Streptomycin, Chloramphenicol or Tetracycline – response to these is poor. The disease tends to burn out after a year or so.

Social Implications of Venereal Disease

Venereal disease, especially Gonorrhoea and Syphilis, are persisting as relatively common conditions due to the large reservoir of untreated cases. For every case treated there is at least one other person affected and failure of notification along with natural reticence of patients to implicate partners hinders attempts to treat contacts.

The social groups which form the largest untreated pool of carriers are prostitutes and male homosexuals. It has been estimated that 50% of Sydney's prostitutes are carriers of gonorrhoea. Homosexuals may harbour syphilis as rectal chancres which are often not even looked for. Similarly, the asymptomatic female carrier of a venereal disease will not be diagnosed unless implicated by a male partner. A possible solution lies in supervised prostitution with enforcement of hygienic conditions. Also more efficient and tenacious investigation could reduce the poor treatment.

3. **Pelvic Infection**

Pelvic infections are those affecting the genital organs above the cervix and the adjacent tissues.

a. *Acute Gonococcal Salpingitis*

Incidence : Gonorrhoea is the most common venereal disease, occurring in 1-2% of Australians. Salpingitis probably occurs in at least 10% of women with lower genital tract (cervical) gonorrhoea but not all of them will present with an acute episode.

Organism : Neisseria gonorrhoea is a Gram-negative diplococcus.

Pathogenesis : This is a venereal disease. The organism, cannot survive in dry conditions.

Pathology : In the female the organism initially infects the mucus-secreting area of the endocervix, and sometimes the urethra, Skene's glands or Bartholin's glands, (it does not penetrate and infect normal squamous vaginal epithelium). Spread may occur from the cervix via the endometrium to the tubal lining which then becomes acutely inflamed. The inflammatory exudate may escape through the ostia or the infection may spread through the muscular wall leading in either case to pelvic peritonitis.

Sequelae :
i. Resolution – this usually occurs following early effective therapy.
ii. Pyosalpinx – this occurs when the fimbrial end of the tube is either adherent to the ovary or closed, causing pus to collect in the tube. Gross destruction within the tube may then occur.
iii. Hydrosalpinx – this occurs when the fimbrial end only is occluded by an infection but the infecting organism is quickly eradicated. Less destruction occurs in the tube.
iv. Fibrosis – often this is associated with multiple adhesions both inside and outside the tube. The latter may involve the bowel. Fibrosis may also involve the ovary, interfering with ovulation.

Infertility occurs in about 50% of women following gonococcal salpingitis.

Symptoms and Signs : As symptoms of gonorrhoea are relatively rare in women females may present only after the male partner develops symptoms. Prior to the development of salpingitis a woman with gonorrhoea may have any of the following:

i. A yellow purulent discharge.
ii. Dysuria.
iii. Reddened cervix covered with exudate.
iv. Reddened urethra; Skene's and Bartholin's ducts thickened and palpable.

If acute salpingitis develops the following symptoms occur with *sudden* onset:

i. Pain across the lower abdomen occurs early and is the usual presenting symptom.
ii. Nausea and vomiting are late symptoms and are less severe than in acute appendicitis.
iii. Pyrexia is severe (up to 39.5° C.), appearing early, with a proportional tachycardia.

iv. Peritonism – abdominal rigidity and involuntary guarding evident across the lower abdomen.
v. Severe pain associated with movement of the cervix, palpation in both lateral fornices, and rectal palpation over the tubes.

Usually no distinct masses can be felt. When these symptoms and signs occur associated with a yellow purulent vaginal discharge, gonococcal salpingitis is suspected.

Differential Diagnosis : i. Other causes of acute salpingitis – post partum and post abortal infection.
ii. Pregnancy states – ectopic gestation.
iii. Other gynaecological conditions:
– Torsion of a tubo-ovarian mass,
– Rupture of an ovarian follicle or cyst.
iv. Others:
– Acute appendicitis.
– Other causes of acute abdomen.

Investigations : Haemoglobin – normal.
White Cell Count – 20-25, 000/mm³ with a polymorphonuclear leucocytosis.
Swab from cervix and urethra (using charcoal impregnated swab and Stuart's transport medium). Direct smear will show intracellular Gramnegative diplococci and culture on chocolate blood agar in CO_2 will show the gonococcal colonies.

Treatment : i. Admit to hospital.
ii. Penicillin G, 2-4 million units every 6 hours for 48 hours with Probenecid 2 gm every 8 hours for 48 hours. Alternatively, tetracyclines can be used orally in doses of 1-2 gm/day for 4-5 days.
iii. Morphine or pethidin once other causes of 'acute abdomen' ehave been excluded.
iv. Laparotomy only if appendicitis cannot be excluded.

b. *Acute Post Partum or Post Abortal Salpingitis*

Incidence : Most common following "amateur-induced abortions.

Organisms and Pathogenesis : Bowel and skin flora (e.g. E. Coli, anaerobic Streptococci, Cl. welchii) invade necrotic retained placental fragments or blood clot. The infection spreads through the myometrium causing a serosal cellulitis and parametritis and may involve the fallopian tubes from the outside. Either type may lead to a state of chronic pelvic inflammation.

Symptoms and Signs : Onset of symptoms is not usually as sudden as with gonococcal salpingitis and there is a recent history of delivery or abortion.
Pyrexia and lower abdominal peritonism occur with a discharge which becomes foul and profuse.

Investigations: Haemoglobin may be low if bleeding has occurred.
White cell count – 15-20,000 /mm 3.
Cervical swab – will grow the causative organism.

Treatment : i. Admit to hospital and confine to bed.
ii. Antibiotics: Penicillin G. 2 million units 6 hourly with either streptomycin or a sulphonamide such as Bactrim. The clinical response after 36-48 hours will indicate whether a change of antibiotic (as indicated by sensitivity results) is needed.
Curettage should be deferred until the inflammation has settled unless there is heavy bleeding.

c. *Chronic Pelvic Infection*

Incidence : Less common since effective treatment of acute infections has been introduced.

Organism : The original bacteria has usually disappeared leaving either sterile pus or a mixed growth of secondary contaminants.

Pathogenesis: Chronic pelvic infection may follow acute

salpingitis or may be associated with tuberculosis or a foreign body. It may lead to any of the sequelae described under acute salpingitis.

Symptoms and Signs : i. A previous history of an acute onset is seldom elicited.
ii. The main complaint is usually pain in the lower abdomen and pelvis often referred to the back. Deep dyspareunia may occur.
iii. Sterility may be the only symptom.
iv. Menorrhagia and disturbances of menstrual rhythm may occur but this is an uncommon symptom.
v. Vaginal discharge is not a feature of chronic infection.
vi. Irritability, depression, anorexia and loss of libido are often present.
vii. On vaginal examination there are usually tender fixed masses on both sides of the pelvis which may extend into the pouch of Douglas. Uterine mobility is reduced. There is no nodularity.

Differential Diagnosis : Pregnancy state – ectopic gestation.
Malignancy – Carcinoma of the ovary, usually is less tender and with a shorter history.
Endometriosis – Pain during menstruation and nodularity in the pelvis is commonly evident. Diverticulitis.

Investigations : Haemoglobin – may be reduced if significant bleeding has occurred.
White cell count – usually normal.
E.S.R. – may be raised.
Cervical swabs – unhelpful.

Treatment : i. Conservative – Bed rest is not usually needed. Pelvic Short Wave Diathermy (under antibiotic cover to avert flare-ups) will accelerate healing.
ii. Surgery – Avoid this approach if possible as the removal of inflammatory tissue may involve total hysterectomy or even complete castration. It can be carried out only in the quiescent phase (3 months or more after infection).
Indications for surgery are:
– Persistent pain not cured by conservative means.
– Recurrent exacerbations.
– If ovarian tumour cannot be excluded.

d. *Pelvic Tuberculosis*

Incidence : This is rare in Australians but is found in 5% of migrant women presenting with infertility.

Pathology : Spread from a pulmonary lesion involves the fallopian tubes initially. Spread to the uterus occurs in 50%, to the ovaries in 30%, and to the cervix and peritoneum more rarely.

Signs and Symptoms : i. Usually detected during infertility investigations.
ii. May present as adnexal inflammatory masses.
iii. Cervical ulcers may mimic malignancy.
iv. Menstrual disturbances may rarely occur.

Investigations : Haemoglobin – may be low.
E.S.R. – usually raised.
Mantoux test – Negative result may disprove the diagnosis.
Chest X-ray – may show inactive or active lesion. Hysterosalpingogram – should not be used if the diagnosis is suspected, due to the risk of dissemination or exacerbation.
Curettage – in the pre-menstrual stage, for histology, culture and guinea pig innoculation.

Treatment : i. Antibiotics. If infertile with no mass, use PAS (or ethambutol) INAH and Streptomycin – or other anti-tuberculous drugs.
ii. Surgery. Total hysterectomy and sal-

pingectomy usually with oophorectomy, is indicated for :
- Adnexal masses,
- Symptoms, e.g. abdominal pain or menorrhagia.
- Failure of medical treatment to achieve cure, as shown by repeated curettage.

Prognosis : Cure of tuberculosis is usual 20% of those without tubal masses treated conservatively will conceive again but only 25% of these pregnancies will reach viability.

Investigation and Diagnosis of Vaginal Discharge and Pelvic Infection

When a woman presents with a vaginal discharge and/or evidence of pelvic inflammation a systematic diagnostic approach is essential. Two things in particular must always be excluded. Firstly, a pregnancy state (including ectopic pregnancy, threatened abortion and hydatidiform mole) and, secondly, malignancy (ovarian to vulval).

1. Take a full gynaecological history and perform a complete examination. This will often give a definitive diagnosis or will at least suggest the relevant investigations to be carried out.
2. Take a Papanicolan smear. This should be done routinely if one has not been taken in the previous twelve months and is particularly indicated in cases of abnormal vaginal bleeding, dyspareunia, or if the cervical epithelium appears abnormal when visualised.
3. If an infective vaginal discharge is present, high vaginal, cervical and urethral swabs should be taken for culturing. If charcoal impregnated swabs and Stuart's transport medium are used, then gonococcii, if present, will survive and grow as distinct colonies. The specimens should be cultured on both blood agar and chocolate blood agar in a CO_2 enriched atmosphere (for gonococci).
4. Microscope slide preparations may be made using gram stains (looking for intracellular gonococci and monilial hyphae) or, if it is desired, trichomonads may be visualised alive in a drop of warm saline.
5. If pelvic infection is suspected, the following investigations may also be helpful.
   . Haemoglobin – may be low if chronic bleeding has occurred.
   . White Cell Count – high values indicate infective processes.

   . E.S.R. – Elevated values are of little use as they may reflect an inflammatory or malignant process.
   . Mantoux test – a negative result may exclude T.B.
   . Chest X-ray – may reveal a primary T.B. focus or a secondary neoplastic lesion.

N.B. D. & C. and Hysterosalpingogram should not be performed if active pelvic inflammatory disease is suspected.

CHAPTER 14

PROLAPSE AND UTERINE DISPLACEMENT

General Instructional Objective

Recognises genital prolapse and uterine displacement and understands the principles of management so that correct advice is given to patients.

Specific Behaviours

1. Describes the pathological anatomy of prolapse.
2. Describes degrees of prolapse.
3. Discusses symptoms of prolapse.
4. Demonstrates an ability to assess women with a genital prolapse.
5. Discusses the management of prolapse.
6. Discusses aetiological factors in a case of prolapse.
7. Demonstrates ability to counsel women with genital prolapse or uterine displacement.
8. Discusses the clinical features of uterine displacement.
9. Performs a vaginal examination and evaluates accurately the position of the uterus.
10. Explains the indications for treatment of uterine displacement.

Prolapse and Uterine Displacement

Prolapse is the falling down, or sinking of a part or viscus.

Pathological Anatomy

Vaginal prolapse may occur without uterine prolapse, but descent of the uterus always carries some part of the upper vagina with it.

1. **Vaginal Prolapse:**

Anterior Wall –

Prolapsing upper part of the anterior wall of the vagina may carry with it the base of the bladder resulting in a cystocele (Fig. 14.1) Prolapse of the weakened lower part of the anterior vaginal wall will result in a urethrocele (Fig. 14.2). The urethra is not dilated but is displaced downwards and backwards.

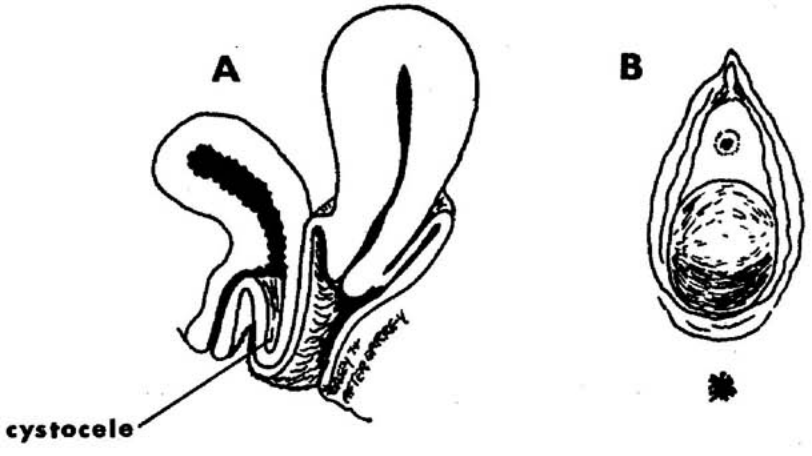

Fig. 14.1.
A. Cross-sectional view of a cystocele. The base of the bladder has prolapsed together with the upper anterior vaginal wall.
B. The anterior vaginal wall is seen bulging through the introitus.

Posterior Wall –

An enterocele forms when the upper part of the posterior vaginal wall herniates into the vagina and carries with it loops of bowels (Fig. 14.3).

When the lower posterior vaginal wall protrudes into the vagina bringing with it the rectum, a rectocele is present (Fig. 14.3).

2. **Uterovaginal Prolapse:**

The prolapsing uterus carries with it the upper vagina. It may also be associated with an enterocele, cystocele or a rectocele (Fig. 14.4). Three *degrees of prolapse* are recognised –

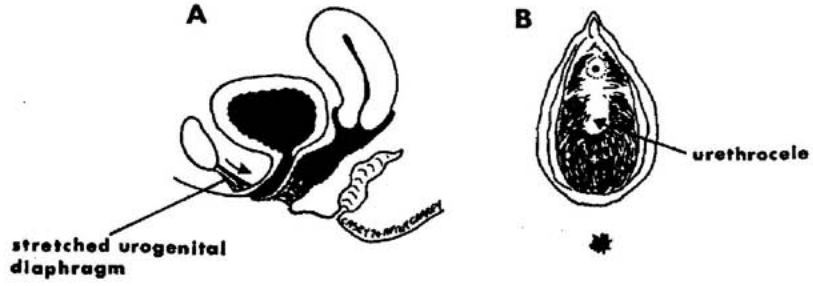

Fig. 14.2.
A. Backward and downward displacement of the urethra on cross-sectional view.
B. A midline urethral bulge of the urethrocele is seen deep to the anterior vaginal wall.

- *First* Slight descent of the uterus with the cervix remaining within the vagina at all times.
- *Second* – Cervix projects through the vulva on straining.
- *Third* – The entire uterus prolapses outside the vulva. It is also called procidentia. Procidentia and prolapse are synonymous but the former is used more often exclusively for third degree descent of the uterus.

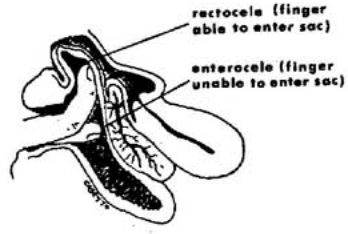

Fig. 14.3. Cross-sectional view of a rectocele and enterocele. Note that the enterocele contains loops of bowel.

Aetiology of Prolpase:

Vaginal and uterine prolapse has a common aetiological background of relaxation of fascial supports. Relaxation of supports may be –

1. Congenital – Rarely a prolapse is seen at birth.
2. Developmental Weakness – this may be familial.

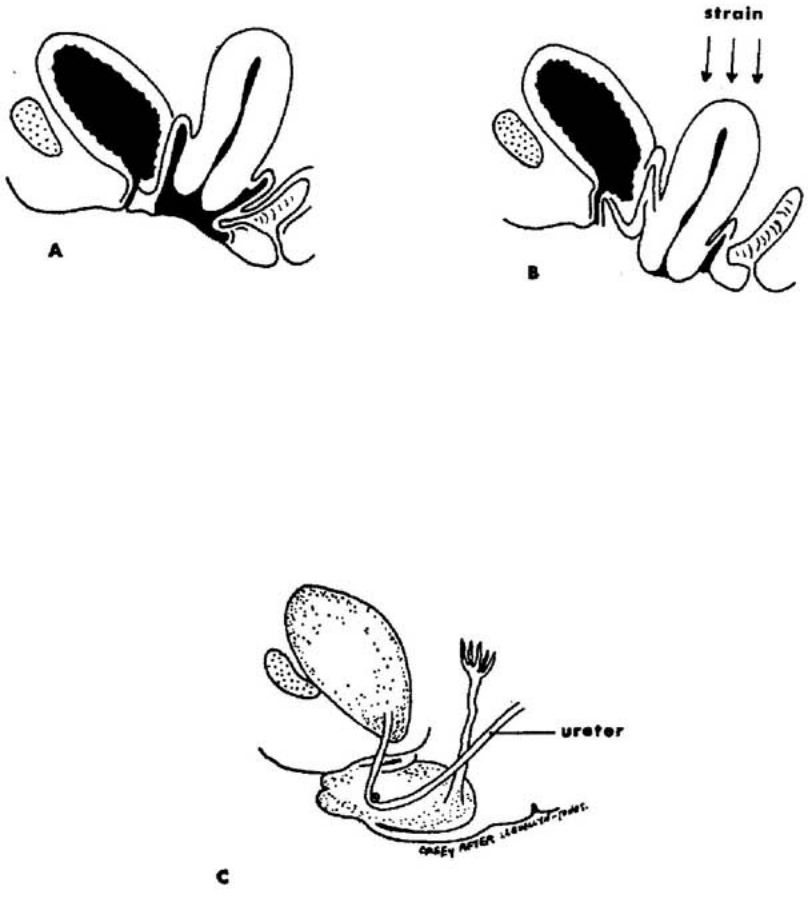

Fig. 14.4. Degrees of uterovaginal prolapse.
A. First degree.
B. Second degree.
C. Third degree or procidentia.
Note there is an associated cystocele in A and B.

3. A result of injury or damage during childbirth. This is probably a major factor. The damage is greatest when:

   a. expulsive attempts are made by the mother before the cervix is fully dilated, stretching the transverse and uterosacral ligaments (Fig. 14.5),
   b. traction is applied to the presenting part before full dilatation of the cervix,
   c. downward fundal pressure is exerted during placental delivery,
   d. the foetal head stretches the vaginal walls during descent (enterocele, rectocele),
   e. the pubovesicocervical fascia is stretched during childbirth allowing the protrusion of the urethra down from under the symphysis pubis (urethrocele, cystocele).
4. The result of atrophy of tissues at the climacteric due to oestrogen lack.

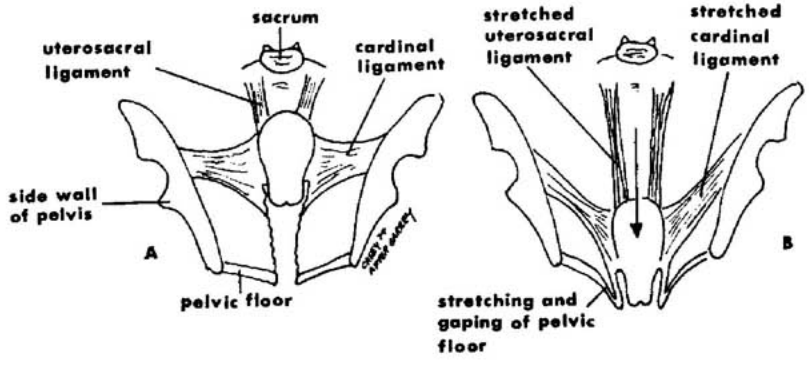

Fig. 14.5.
A. Normal ligamentous supports of the uterus.
B. The mechanisms of the uterovaginal prolapse showing stretched uterine supports.

Symptoms of Prolapse:

Only a few of the following symptoms may be experienced by a particular patient. Characteristically, most symptoms can be relieved on lying down.

1. Sensation of "something coming down" in the vagina. This may be the bulge due to the uterine prolapse, enterocele, cystocele or rectocele.

2. "Bearing down" feeling. This may be due to the pressure of the mass on the rectum creating a desire to defaecate.
3. Urinary symptoms – These depend on the descent of the anterior vaginal wall and the displacement of the bladder and urethra.
   a. frequency – due to mechanical and later infective cause.
   b. difficulty in emptying the bladder – due to pooling in the cystocele.
   c. stress incontinence – not necessarily a symptom. Present when the posterior urethrovesical angle has been lost, or sphincter mechanism damaged.

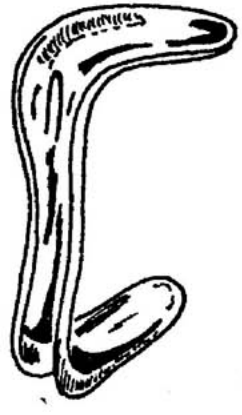

Fig. 14.6. Sim's speculum.

4. Difficulty in emptying the rectum – here faeces are collecting in the rectocele.
5. Backache – midline at the lumbosacral level, diffuse, deep seated, and accompanied by tenderness. Completely relieved immediately on resting. It is more common in uterine prolapse where uterosacral ligaments are stretched.
6. Dyspareunia or apareunia due to malposition of organs or dilatation of vaginal introitus.

Signs of Prolapse:

1. **Uterovaginal Prolapse**

   Even minor degrees of prolapse may be recognized by feeling the cervix while the patient is straining, or by pulling on the cervix with vulsellum forceps. If there is doubt the patient should be asked to stand or walk for some time before the examination.

2. **Vaginal Prolapse**

   After the insertion of a Sim's speculum (Fig. 14.6) the patient is

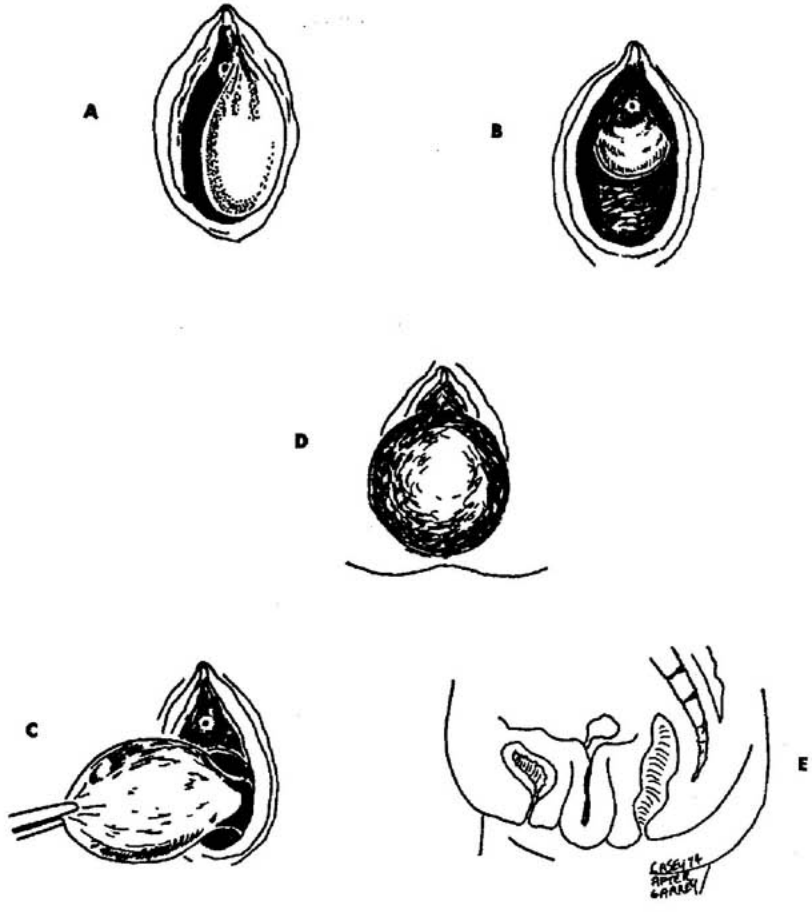

Fig. 14.7. Differential diagnosis of prolapse.
A. Cyst of Bartholin's gland.
B. Cyst of Skene's duct.
C. Vaginal cyst.
D. Cervical polyp.
E. Chronic inversion of the uterus.

asked to bear down or cough. The anterior and posterior walls are observed systematically. Differentiation between a recotcele and enterocele may be made on a rectal examination (Fig. 14.3). Stress incontinence is brought on by asking the patient to cough. A trickle of urine may be seen emerging from the urinary meatus.

Differential Diagnosis of Prolapse (Fig. 14.7):

Prolapse may be mimicked by the following:

1. Cyst of Bartholin's Gland.
2. Cyst of Skene's duct.
3. Vaginal cyst, e.g. of Wollfian duct origin.
4. Cervical polyp.
5. Chronic inversion of the uterus.

Complications of Prolapse:

1. Keratinization of prolapsed vagina – vaginal walls become thick and hard.
2. Ulceration – especially with proccidentia (decubitus ulcers).
3. Obstruction of the urinary tract – difficulty in emptying the bladder results in hypertrophy of the bladder and trabeculation. Angulation of the lower ends of the ureters causes attenuation, constriction, and eventually hydroureter, hydronephrosis, urinary tract infection and renal involvement.
4. Hypertrophy of the cervix – this is probably to a large extent due to oedema and congestion.
5. Incarceration of the prolapse – occurs when the proccidentia become oedematous and difficult to reduce.

Management:

1. *Prevention*

   This is carried out during childbirth, by –

   a. avoidance by patient of pushing before full dilatation of the cervix,

   b. avoidance of a long second stage of labour by use of generous episiotomy or forceps delivery,

   c. avoidance of fundal pressure in the third stage of labour.

   d. early puerperal ambulation and pelvic floor exercises.

2. **Active Treatment**

   Active treatment may be conservative or operative.

   a. *Conservative*

      Undertaken in a woman who –

      i. refuses operation or wishes to delay it,
      ii. is not fit for surgery,
      iii. has a short life expectancy.

      Treatment is by stretching the vaginal walls supporting the uterus by a plastic ring called a pessary (Fig. 14.8). The pessary is replaced every four months. This treatment relieves symptoms but is not curative. Resulting complications include constipation, urinary incontinence, vaginal ulceration, rarely carcinoma.

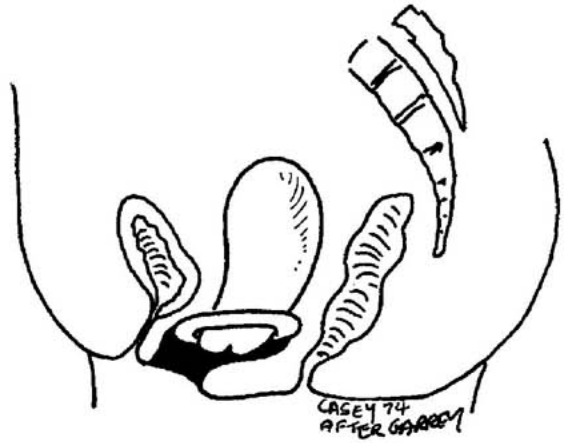

Fig. 14.8. Vaginal pessary in position. Note its potential pressure effect on the urethra and rectum.

   b. *Operative*

      This is the best treatment but is undertaken only if the condition is causing symptoms. Urinary tract infection is treated preoperatively; ulcerated prolapsed parts can be treated with local oestrogens after reduction of the procidentia.

Anterior Colporrhaphy – Designed for urethrocele and cystocele. The pubocervical fascia is stitched together in the midline to support the bladder, and redundant anterior vaginal wall is removed and margins approximated.

Posterior Colpoperineorrhaphy – Used for the rectocele. A triangular portion of the posterior vaginal wall is excised. The rectovaginal fascia is sutured and the levatores ani approximated.

With an enterocele the peritoneum must be entered, the hernial sac excised, vaginal vault repaired, and the uterosacral ligaments approximated to form a roof.

Manchester Operation – For uterovaginal prolapse. The operation consists of anterior colporrhaphy, amputation of the cervix to approximate the cardinal ligaments, and posterior colpoperineorrhaphy.

Vaginal Hysterectomy – This operation is often performed for uterovaginal prolapse, in combination with anterior colporrhaphy and posterior colpoperineorrhaphy, because after menopause, the uterus remains as a nuisance organ that can develop potentially fatal pathology. For this operation to be performed the size of the uterus must be less than that of a 12-14 week pregnancy, if fibroids are present.

Backward Displacement of the Uterus

The normal uterus is in a position of anteversion and anteflexion (Fig. 14.9). Retroversion of the uterus, a term often used to mean both retroversion and retroflexion, is present in about 10% of all women. In an otherwise normal pelvis a mobile, retroverted uterus *usually produces no symptoms*. Symptoms of dysmenorrhoea, menorrhagia, and dyspareunia may occur with a fixed and retroverted uterus, but are probably the result of the lesion fixing the uterus. Retroversion has been implicated in infertility and recurrent abortion but evidence is controversial.

Aetiology:

1. **Developmental**

   In infancy the uterus is retroverted, and in a percentage of women it will not attain its anteverted position with the growth of the corpus.

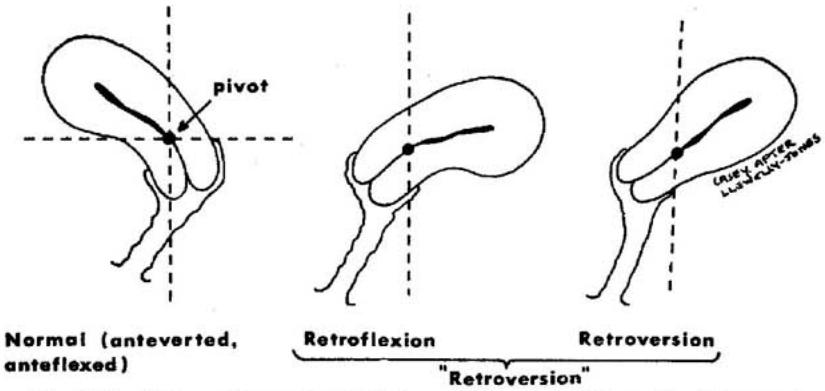

Fig. 14.9. Retroversion and retroflexion of the uterus. The axis of the cervix determines the version, while the axis of the body of the uterus determines the flexion.

2. **Acquired**

   a. *Puerperal* – during involution after pregnancy the round ligaments remain slack.

   b. *Tumours* in front of the uterus. Fibroids are the most common pathological tumours found. A distended bladder may simulate a tumour.

   c. *Endometriosis* of uterosacral ligaments, shortening them.

   d. *Adhesions* after pelvic infectons.

Diagnosis:

On speculum inspection the cervix will be pointing forward. On bimanual examination the body of the uterus will be felt in the pouch of Douglas.

Management:

In a patient without symptoms *no treatment* is offered. When symptoms are present treatment may be by a *Hodge Pessary* (Fig. 14.10); or by surgery. The pessary is indicated with a mobile uterus where doubt exists as to the cause of symptoms and a Pessary Test can be attempted. Here the pessary is placed in position (Fig. 14.11) for one month after which the symptoms are reviewed. If improvement is experienced the pessary is removed for another month but now the uterus is retroverted without the patient's knowledge. If symptoms return surgery will probably be beneficial.

The pessary is also indicated in cases of infertility and recurrent abortion where other causes are excluded; in a pregnancy where the uterus does not correct itself by the 10th to 12th week; and where the retroversion is causing symptoms but surgery is refused.

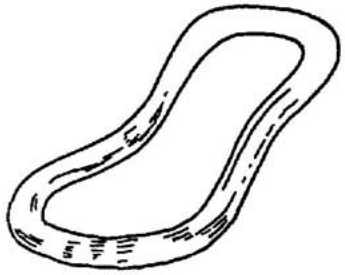

Fig. 14.10. The Hodge Pessary.

Surgery is rarely necessary and is carried out only after a positive Pessary Test. The technique may involve ventrosuspension with shortening of the round ligaments (Gilliam's Operation), although numerous procedures have been described.

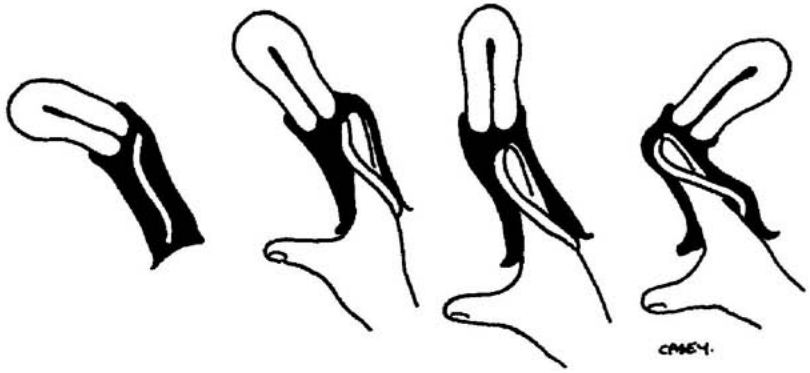

Fig. 14.11. Correction of retroversion by means of a Hodge pessary.

Retroverted Gravid Uterus

A pregnancy occurring in a retroverted uterus usually rises out of the pelvis spontaneously. Rarely, mechanical impaction deep to the sacral promontory can take place at 12 to 14 weeks. The uterus then expands and the cervix then displaces the bladder upwards, elongating

the urethra. The patient may then present with acute retention of urine. On examination the bladder is the soft abdominal swelling, and vaginally the cervix is difficult to locate, being high up in the anterior fornix.

Treatment is by slow catheterization of the bladder and disimpaction of the uterus. If this does not cure the impaction the uterus is manually anteverted under anaesthetic and a pessary inserted to maintain anteversion.

Inversion of the Utherus

In this condition the uterus is turned inside out through the cervix. This may vary in degree (Fig. 14.12). Acute inversion usually occurs after the third stage of labour (see page 9.6).

Chronic Inversion

This may be puerperal, being discovered weeks to months after delivery but having its origin at the delivery of the placenta. Rarely uterine attempts to expel an intracavity fibroid can cause inversion. The patient complains of irregular bleeding and a sensation of "something coming down".

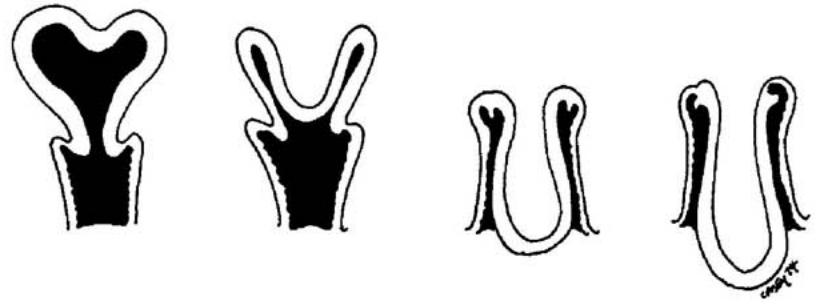

Fig. 14.12. Degrees of uterine inversion.

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CHAPTER 15

DYSMENORRHOEA, DYSPAREUNIA, ENDOMETRIOSIS, ADENOMYOSIS PELVIC INFECTION "OR UTERINE PROLAPSE"

General Instructional Objective

Comprehends the patho-physiology of pelvic discomfort in non-pregnant women so that adequate investigation, support or management of the condition can be achieved.

Specific Behaviours

1. Lists the common causes of dysmenorrhoea, dyspareunia and pelvic pain.
2. Demonstrates how a correct diagnosis is achieved in a patient with pelvic discomfort.
3. Explains how certain relevant pelvic conditions can cause specific pelvic discomforts.
4. Prescribes a course of management which will alleviate the symptoms of a woman with pelvic discomfort.
5. Specifies what investigatory steps should be taken to achieve a diagnosis in a woman with pelvic discomfort.
6. Differentiates between congestive dysmenorrhoea and spasmodic dysmenorrhoea by history and physical findings.
7. Describes the physical findings in conditions which may cause pelvic pain.
8. Counsels a woman with pelvic discomfort.

Pelvic Pain – Dysmenorrhoea, Dyspareunia

(including Endometriosis and Adenomyosis)

A. **Introduction** – causes, characteristics and investigation of pelvic pain.

B. **The causes of dysmenorrhoea and dyspareunia.**

C. **The aetiology and other factors associated with dysmenorrhoea and dyspareunia.**

D. **Clinical presentation and diagnosis of main causes of dysmenorrhoea.**

E. **Management of the causes of dysmenorrhoea and dyspareunia.**

F. **Counselling a patient with pelvic discomfort.**

A. Introduction

Pelvic pain is a common symptom. It may be associated with organic or functional disorders of –

1. the genital system – e.g., pelvic infections, tumours of the uterus.
2. the gastrointestinal system – diverticulitis, carcinoma.
3. the urinary system – infection (cystitis), calculi.
4. the musculoskeletal system – muscle strain, oesteoarthritis, structural disorders (e.g., kyphosis, scoliosis, disc lesions, metastatic tumours.)
5. and nerves.

In addition, psychogenic pain is not uncommon.
In gynaecology the most common types of pelvic pain are those associated with:

a. dysmenorrhoea – painful menstruation, and
b. dyspareunia – difficult or painful coitus,

and are detailed in a following section.
Pelvic pain may be of various types and a careful history must be elicited:

1. **Acute**
   a. colicy or cramp-like
   b. of sudden onset
   c. sharp and clearly defined
   d. tender on examination, e.g. inflammation, torsion of a pedunculated tumour, tubal pregnancy.

2. **Chronic**
   a. gradual onset,
   b. dull or dragging,
   c. poorly defined,
   d. deep-seated, (related to pressure or traction on ligamens or where other viscera are involved or due to congestion,)
   e.g. . Tumours of the uterus or ovaries (including carcinoma.)
   . Prolapse.

3. **Acute and Chronic** – notably in endometriosis.

4. **Suprapubic** –
   e.g. . Salpingitis.
   . Cellulitis
   . Many other abdominal causes.

5. **Sacral backache**
   a. continuous ache,
   b. unaffected by movement (as opposed to "orthopaedic" backache), e.g.
   . Chronic pelvic inflamation.
   . Endometriosis.
   . Uterine prolapse.

6. **Associated factors**
   a. relationship to menstruation or coitus,
   b. other features of, e.g. urinary tract infection such as frequency, relationship to movement, lifting, constipation, etc.

Following a detailed history and both general and pelvic examination (see Chapter 1) it may be necessary to carry out some of the following investigations:

i. Laboratory tests for systemic disease, e.g., urinary tract infection, syphilis, blood dyscrasia, porphyria.

ii. X-rays of pelvis, spine, gastrointestinal tract (including gallbladder).

iii. Sigmoidoscopy.

iv. Examination under anaesthetic.

v. Cystoscopy and pyelography.

vi. Culdoscopy.

vii. Determination of ovulation.

viii. Laparoscopy or laparotomy.

ix. Psychiatric evaluation.

In diagnosing the cause of pelvic pain the first step is to exclude disturbances of other systems. Pain related to menstruation or coitus is of gynaecological importance. This does *not* mean that conditions causing dysmenorrhoea or dyspareunia cannot cause pain at other times, e.g. chronic pelvic inflammatory disease, but that these symptoms highlight gynaecological disturbances.

In the following sections the causes, aetiology, variation of clinical presentation and management of conditions causing pelvic discomfort – manifested as dysmenorrhoea and dyspareunia – are outlined, so that direct comparisons can be made at each level.

B. The causes of Dysmenorrhoea and Dyspareunia

1. Dysmenorrhoea:

a. *Primary or spasmodic dysmenorrhoea* – first day dysmenorrhoea associated with ovulatory cycles. Also referred to as physiological, functional or idiopathic dysmenorrhoea.

b. *Secondary dysmenorrhoea* –

i. Congestive or premenstrual dysmenorrhoea due to pelvic infection.

ii. Endometriosis and adenomyosis.

iii. Ovarian.

c. *Membranous dysmenorrhoea* (rare).

2. Dyspareunia: – may be subdivided as to –

a. *Primary* – manifest during initial attempts at intercourse.

b. *Secondary* – appearing after a period of normal, painless intercourse (usually some years later). It frequently follows the birth of a child.

c. *Superficial* – when pain or difficulty occurs during penetration.

d. *Deep* – when it occurs after penetration.

On this basis the causes of dyspareunia may be classified as in Table 15.1.

	Superficial	Deep
Primary	**Vaginismus due to–** faulty sex education ignorance fear initial clumsy attempts at coitus past psychological trauma **Rigid hymen** **Atresia** of vaginal orifice	Vaginal obstructions, hypoplasia, vaginal septum Pelvic tumours
Secondary	Vaginismus Local trauma Inflammation– vulvitis, Bartholinitis Urethral conditions Scarring and contracture due to obstetrical injuries and gynaecological operations Anal conditions	Infections– vaginitis, salpingo-oophoritis Endometriosis Retroverted uterus Prolapsed ovary Pelvic tumours Tender bowel lesions "Cervical dyspareunia" Psychosomatic

Table 15 – Causes of dyspareunia.

NOTE: **Apareunia** – inability to practise coitus. This differs from dyspareunia mainly in degree.

C. Aetiology and other factors (of dysmenorrhoea and dyspareunia):

1. Primary dysmenorrhoea

During the menstrual cycle the activity of the myometrium is governed first by oestradiol and then progesterone (following ovulation). These hormones produce contractions of high frequency low amplitude and lower frequency, high amplitude respectively.

At the end of the cycle prostaglandin $F_{2\alpha}$ is liberated as the endometrium fragments. This substance produces very large contractions on the first day of menstruation, and like those contractions occurring during labour, they attempt to produce cervical dilatation, which gives rise to pain. In some people nausea, and even vomiting, occurs as a result of stimulation of the sympathetic nervous system. After the first day of menstruation the uterine contractions gradually diminish in amplitude and by the end of menstruation they have returned to the pattern produced by oestradiol.

With failure of ovulation, there is no secretary endometrium formed, less prostaglandin $F_{2\alpha}$ is liberated and no pain occurs. Consequently oestrogen withdrawal bleeding is free from discomfort – as usually occurs in the first few years of menstrual function.

Other theories – *obstructive theory* – the outflow of menstrual blood is obstructed and consequently results in irregular spasmodic and painful contractions.

(Further theories are described in "Postgraduate Obstetrics and Gynaecology", 4th Ed., J.C. McClure Browne, pp 89-92).

Incidence:

At least 50% of women experience some dysmenorrhoea and 10% (between the ages of 18 and 24 years) seek medical advice. Up to 5% are incapacitated for a short while.

2. **Secondary Dysmenorrhoea**

a. *Congestive dysmenorrhoea*

Sodium is lost from the renal tubules as progesterone antagonises the action of aldosterone. Consequently the renin-angiotensin system is stimulated to produce more aldosterone in order to conserve sodium for physiological needs.

Progesterone concentration falls at the end of the cycle and leaves an excess level of aldosterone resulting in temporary salt and water retention. These normal changes are accentuated by the increased vascular permeability or venous congestion associated with pelvic inflammation. The increased tissue pressure stimulates nerve especially in the ovaries.

b. *Endometriosis* (or extrauterine endometriosis) is the extrauterine growth of tissue having the structural and functional properties of endometrium.

There are three main theories postulated as causing the lesions:

i. *Sampson's endometrial spill theory* (1921) – during menstruation endometrial material passes out through the tubes, spills into the peritoneal cavity, implants and grows.
Evidence: menstrual debris has been grown in tissue culture explains lesions in peritoneum and scars but not in umbilicus, chest or limbs.

ii. *Serosal cell metaplasia.* (Meyer's and Ivanoff's theory) – Coelomic cells forming the Müllerian ducts are thought to retain the capacity to differentiate into endometrium and myometrium.

Explains peritoneal lesions.

iii. *Lymphatic and vascular embolism* (Halken, 1925) of endometrial fragments.

Explains distant lesions.

It is likely that all three of these hypotheses may act to explain all the many types of endometriosis.

Pathology:

. "Chocolate" (blood-filled) cysts lined by columnar epithelium and surrounded by endometrial stroma.

. Fibrous reaction of the affected tissues produces a cystic tumour.

Sites:

i. *The ovary* (commonest site)

. chocolate cysts.
. usually bilaterally, but can be unilateral,
. usually no larger than 5cms. (but may fill the pelvis),
. accompanied by dense adhesions and often causes infertility.

ii. *Round ligaments, uterosacral ligaments* – painful nodules in the latter are almost diagnostic of endometriosis.

iii. *Fallopian tubes* (very rare site)

iv. *Rectovaginal Septum*

v. *The Intestines* may be involved causing bowel obstruction

vi. *Bladder and ureters*

viii. *Abdominal wall* (the umbilicus and scars from abdominal surgery)

viii. *Lungs, limbs, perineum.*

3. In *dyspareunia* the specific causes and aetiology are closely allied, e.g. dyspareunia of mechanical psychological or educational background. Again, any inflamed tissue is naturally painful.

D. Clinical Presentation and Diagnosis of Main Causes of Dysmenorrhoea

1. **Primary dysmenorrhoea** (Of uterine origin; directly due to menstruation) **History:**

i. *Age* – symptoms start usually several years after menarche, although some girls experience painful periods from the outset.

ii. *Parity* – usually young nulliparous women; pain is often relieved by the first pregnancy, due to softening of the cervix, and easier escape of menstrual flow.

iii. *Pain* – cramp-like colicky, maximal on first day of menstrual flow, may be mild on second day, referred to the T_{10}, L_1 dermotomes.

iv. *Associated symptoms* – nausea and vomiting, fainting may occur. *On examination* – bimanual examination reveals no abnormality.

2. **Secondary dysmenorrhoea**

a. *Congestive dysmenorrhoea:*

History–

i. discomfort starts prior to period,

ii. pain relieved by onset of bleeding,

iii. often a diffuse dull ache in pelvis,

iv. often backache,

v. usually history of pelvic infection (e.g. following miscarriage) and often patient has not been completely free of symptoms,

vi. often associated menorrhagia.

On examination – there may be evidence of chronic pelvic inflammation (see Chapter 13).

Doubtful cases – may be resolved by laparoscopy.

b. *Endometriosis:*

History–

i. *Age* – childbearing age, especially 25-35 years.

ii. *Socioeconomic background* – more common in higher social groups (later marriage, pregnancy).

iii. *Parity* – 50-70% nulliparous

40% with history of infertility (tubal adhesions or ovarian dysfunction).

iv. *Dysmenorrhoea–*

. increases in severity 2-3 days premenstrually,
. most severe on first and second days of menstruation (heaviest flow),
. continues to days 3 and 4,
. symptoms begin about 25 years of age,
. signs classically appear after 30 years of age and gradually increase in severity,
. site depends on site of lesion, e.g. lower abdomen, pelvis, rectum and sacrally.

v. *Abnormal menstruation–*

. not common, about 20-25% patients,
. may be excessive or irregular bleeding.

vi. *Dyspareunia–*

. in about 50% (fixed retroversion, lesions in pouch of Douglas or rectovaginal septum).

vii. *Associated symptoms–*

. many and varied, depending on site of lesions,
. includes intestinal or ureteric obstruction, frequency or haematuria,
. general malaise,
. may suggest acute appendicitis when a blood cyst bursts.

On examination–

. nodules in uterosacral ligaments are most suggestive of the diagnosis,
. fixed retroversion.

NOTE: There is a complete clinical spectrum to endometriosis – it may be asymptomatic, even when quite extensive, or may be a painful, sterilising, crippling condition.

Differential Diagnosis–

i. Chronic salpingo-oophoritis

ii. Uterine fibromyomata

iii. Malignant disease–

- ovary,
- peritoneum,
- uterus,
- cervix.

iv. Acute abdominal conditions

v. Causes of haematuria

vi. Diverticulitis

Investigations–

i. Culdoscopy

ii. Sigmoidoscopy – if bowel thought to be involved.

iii. Cystoscopy – if bladder thought to be involved.

iv. Laparotomy – may be necessary for confirmation of the diagnosis.

C. **Adenomyosis:**

Classically presents in older multiparous women and the majority of cases are diagnosed only by the pathologist.

History–

i. *Menorrhagia* in 85% of cases.

ii. *Dysmenorrhoea* during period (33%)

On examination – symmetrically enlarged uterus (rarely larger than that of twelve weeks' pregnancy).

E. **Management of the causes of dysmenorrhoea and dyspareunia**

1. **Primary Dysmenorrhoea**

a. *Analgesics* – Mild dysmenorrhoea may be relieved by simple analgesics (aspirin, phenacetin, paracetamol, codeine) or a small quantity of alcohol. This is only symptomatic treatment and is inadequate for the more severe cases.

b. *Hormonal Therapy* – Before initiating treatment, it is important to consider–

i. the age of the patient. It is preferable not to give a young girl large amounts of progesterone.

ii. the sensitivity of the patient to hormonal preparations – the longer the cycle the more sensitive the patient,

iii. the presence or absence of menorrhagia,

iv. the possible need for oral contraception in addition to relief of symptoms.

If dysmenorrhoea is not associated with menorrhagia then relief may be achieved by suppressing ovulation with ethinyl oestradiol (EE)–

- EE 0.05 mg. daily starting on the first day of menstruation and given for 26 days,
- EE 0.04 mg. daily for young girls not exposed to the risk of pregnancy,
- EE 0.03 mg. daily may suppress those with long cycles (i.e. greater than 33 days).

These methods will produce a painless withdrawal bleed about five days after the last oestrogen tablet. However, after one or two cycles the menstrual loss may become prolonged and sometimes heavy, with a variable time of onset.

Cycle control can be improved by giving a weak progestogen, such as norethynodrel 1 mg. daily, from the 20th to 26th days of the cycle with the ethinyl oestradiol. Withdrawal bleeding commences two days after the last dose of progestogen and is similar to that encountered with normal menstruation.

Sometimes this menstrual loss is on the heavy side. Under these circumstances the quantity of blood lost can be reduced by starting the norethynodrel on the 18th day or by using a more potent progestogen such as norethisterone 0.4 mg. from the 20th day of the cycle.

Norethisterone, while effective in reducing menstrual flow, frequently stimulates myometrial activity sufficiently to produce dysmenorrhoea in susceptible subjects. Some women cannot tolerate 0.4 mg. norethisterone for more than four or five days without experiencing discomfort with menstruation.

When dysmenorrhoea is associated with menorrhagia pain relief is often obtained by reducing menstrual flow. This can be accomplished by using norethistrone from the fifth day of the cycle. This type of therapy is indicated in cases that experience maximum flow and more severe pain on the second day of menstruation.

Oral contraceptives may be used to relieve moderate degrees of dysmenorrhoea. Sequential or combined types of oral contraceptives are effective. In the latter, oestrogen stimulates the regeneration and growth of the endometrium, but when the progestogen is given as early as the fifth day, the effect of oestrogen is inhibited before much growth of the endometrium has occurred. The reservoir of prostaglandin in the endometrium is small and the quantity released at the end of the cycle is insufficient to produce myometrial contractions large enough to produce dysmenorrhoea.

c. *Prostaglandin Antagonists*

Indocid may be used, in conjunction with above measures, in refractory cases.

d. *Operative management*

i. *Dilatation of the cervix* is successful in about 33% of cases. However, there is risk of an incompetent os in subsequent pregnancies.

ii. *Injection of sacral plexus* with alcohol.

iii. Pre-sacral neurectomy-involves laparotomy and if some of the sensory fibres are missed, relief will not be complete.

2. **Secondary Dysmenorrhoea**

a. *Congestive dysmenorrhoea*

i. *Diuretics* may be given towards the end of the cycle to correct salt and water retention. Relief may be incomplete and polyuria is a nuisance.

ii. *Hormonal therapy* – similar to that for primary dysmenorrhoea may be useful as progesterone production is suppressed.

iii. Specific *antibiotic therapy* is indicated for pelvic infection.

iv. Surgery to remove affected lesions may be indicated such as for adenomyosis or salpingitis.

b. *Endometriosis*

Medical Management:

If the symptoms are not severe analgesics may be sufficient, especially if there is likelihood of the woman becoming pregnant as this will induce a natural remission. Control may be achieved hormonally by–

i. producing a *pseudo pregnancy* for six or nine months, or

ii. a pseudo *menopause* can be produced by using a potent progestogen intermittently for three weeks every lunar month to achieve a thin atrophic endometrium, both in the uterus and in the endometrial deposits which bleeds very little at the end of each course of tablets.

The psuedo pregnancy can be produced by starting with ethinyl oestrodiol 0.05 mg. for two weeks and then adding progestogen; either a nor-testosterone or a non-androgenic type of progestogen can be used. The latter has the advantage of producing less stimulus to appetite, so weight gain in overweight women is not as marked. When this progestogen is added, ethinyl oestradiol should be increased to 0.1 mg daily.

To prevent breakthrough bleeding, the dose of oestrogen will need to be increased in a further four weeks. With further increments in the amounts of oestrogen, the dose of progestogen can be increased, but a high oestrogen to progestogen ratio should be maintained to prevent breakthrough bleeding. The object of the hormonal treatment is to transform all endometrium both in ectopic sites and in the uterus, to decidua, with the expectation that, when the hormonal therapy is withdrawn, the decidua will be shed. The advantage of the pseudo-pregnancy approach is that it can also be used in an endeavour to improve fertility in the less chronic cases, who have not developed secondary adhesions due to the escape of blood from the cholcolate cysts.

The disadvantages of this therapy are –

i. Side effects of the drugs.
ii. Fibroids may enlarge greatly.
iii. Breasts may become enlarged and tender.
iv. Results may be temporary.

A pseudo-menopausal condition can be produced by giving norethisterone 0.5 mg. or 1 mg. daily combined with ethinyl oestradiol 0.05 mg. or as a reversed sequential formulation in which the ethinyl oestradiol is added only after the first ten days of progestogen therapy. A week without active medication intervenes, between each three week course of active therapy. If, however, less frequent vaginal bleeding is acceptable, each course of active therapy can be continued for four or five weeks. However, breakthrough bleeding may occur more commonly under these circumstances.

Surgical Management

i. *Conservative* – i.e. removal of areas of endometriosis but leaving reproductive function intact. Urgent symptoms may indicate surgery, e.g. ruptured chocolate cyst.

ii. *Radical* – Removal of ovaries should be carried out when symptoms are very severe, when bladder or bowel is involved, and when reproductive function is no longer a problem. This management cures the condition.

Radiotherapy

i. *Conservative* – i.e. temporary suppression of ovarian function, is almost never used.

ii. *Castrating* – ablation of ovarian function only where surgery is contraindicated.

c. *Adenomyosis*

Management involves either relieving the symptoms (sedation, reassurance or hormonal therapy) or removing the lesions, which usually means hysterectomy if the symptoms are severe enough.

d. *Ovarian dysmenorrhoea*

If one ovary is involved by a pathological process then pain may be felt in that iliac fossa only. Treatment involves those measures described for congestive dysmenorrhoea except if gross enlargement is present when surgical treatment may be necessary.

3. **Primary Dyspareunia**

a. *Vaginismus* – usually stems from a combination of the factors mentioned. There is voluntary contraction or spasm of the muscles of the vaginal introitus and lower third of the vagina, and adductor muscles of the thigh.

Treatment depends largely upon listening sympathetically before proceeding to a general physical examination which concludes with a gentle pelvic examination, using at first one finger and later two fingers. If no mechanical barrier to coitus exists the patient must be reassured as to her capacity for coitus and be given adequate instruction in sex technique. Ideally the husband should be interviewed and the situation explained to him.

Careful patient follow-up is essential. Continued reassurance should be given as to her being anatomically normal and that cure is certain. Pelvic examination should be performed at each visit until three fingers are able to be inserted without difficulty into the vagina. The object is to encourage the patient to gain confidence in herself.

In more difficult cases, initial examination under anaesthetic is followed up by the insertion of graduated vaginal dilators – at first by the doctor and subsequently by the patient herself. It has been significantly stated that in reality you are dilating the patient's mind.

b. *Rigid hymen* – radial incision or excision of hymen.

c. *Atresia or stenosis of vaginal orifice* – perineotomy may be required (e.g. Fenton's operation).

d. *Vaginal septum* – excision.

e. *Pelvic tumours* – see Chapters 18 and 19.

4. **Secondary Dyspareunia**

a. *Inflammation* – appropriate antibiotics and in the case of Bartholin's abscess – marsupialization, senile vaginitis – oestrogen

(ethinyl oestradiol 0.01 mg. daily). Salpingo-oophoritis – see Chapter 13.

b. *Scarring and Contracture* – as many as 20% of women having repair operations subsequently suffer dyspareunia because the vagina is too narrow (Francis and Jeffcoate, 1961). If the patient is having, or is likely to be having, intercourse in the future a colpo-perineorraphy may be considered. Usually a clear indication for this operation, e.g. a symptomatic rectocoele, should be present. At the end of the operation the vagina should admit three fingers.

c. *Endometriosis* – already discussed.

d. *Retroverted uterus* – ventrosuspension.

Prolapsed ovaries – anatomical restoration. Some authorities recommend a change in coital posture rather than surgical intervention for these conditions.

e. *Urethral conditions*–

i. *Urinary Tract Infection* – treat with appropriate antibiotics, e.g. sulfonamides or nitrofurantoin

ii. *Urethral syndrome* – irritation of the urethra by frequent coitus may lead to cystalgia with pain and frequency of micturition soon after coitus. Consequently the patient may avoid coitus. Treatment includes antibiotics (if infection is found) or urinary tract analgesics.

f. *Psychosomatic* – deep dyspareunia occurring in the hours following coitus.

F. Counselling a woman with pelvic pain

A detailed history should allow a confident diagnosis to be made (without further investigation) in most cases. The cause is explained to the patient *in terms that she will understand*, and if further investigations are required, their purpose is also explained.

Explanation or reassurance may form one of the cornerstones of management as in primary dyspareunia, or may be necessary in altering management, e.g. in primary dysmenorrhoea.

It is very important to reassure the patient that her discomfort is *not* due to cancer (if this is true), as often it is a very real fear and commonly not voiced.

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CHAPTER 16

INFERTILITY, AMENORRHOEA AND CONTRACEPTION

General Instructional Objective

Understands the basic principles in conception and amenorrhoea so that he may give appropriate advice to patients.

Specific Behaviours

1. Explains the physiology of ovulation and conception.
2. Assesses, by history and examination, the problems of women with infertility and/or amenorrhoea.
3. Competently counsels women with problems of conception.
4. Advises women with amenorrhoea and infertility.
5. Discusses the pharmacology of drugs and the side effects of methods used in conception control.
6. Discusses the aetiology and management of problems of conception or amenorrhoea.

Infertility, Amenorrhoea and contraception

A. Physiology of Ovulation and Conception

Endometrial changes during an ovulatory cycle are governed by variations in ovarian hormones, (oestrogen and progesterone,) whose production is controlled by the gonadotrophins (Follicle Stimulating Hormone,

F.S.H. and Leutinizing Hormone, L.H.) from the anterior pituitary (Fig. 16.2). The anterior pituitary is under the influence of releasing hormones (F.S.H. – Releasing Hormone, and L.H.–R.H.) produced by the hypothalamus, which receives nerve impulses from the higher nerve centres and is affected also by circulating oestrogens, progesterone, gonadotrophins, and melatonin (Fig. 16.2).

Hypothalamus. Here two centres secrete the decapeptides F.S.H.– R.H. and L.H. – R.H. ,which are either identical or closely related, The basal centre regulates basal secretion of gonadotrophins in both sexes. The cyclic center with its endogenous rhythm of hormone release, accounts for the mid-cycle peaks in the female hormonal pattern (Fig. 16.3). The cyclic centre is inhibited by androgens (Fig. 16.2) and therefore does not function in the normal male.

Anterior Pituitary. Picogram (10-12 bgm.) quantities of F.S.H.–R.H. and L.H. – R.H. circulate through the pituitary portal system bathing the basophil beta cells of the adenohypohysis. These in turn release F.S.H. and L.H., which are glycoproteins of a molecular weight of 41,000 and 30,000 respectively. The releasing hormones may influence the secretion of more than one anterior pituitary hormone.

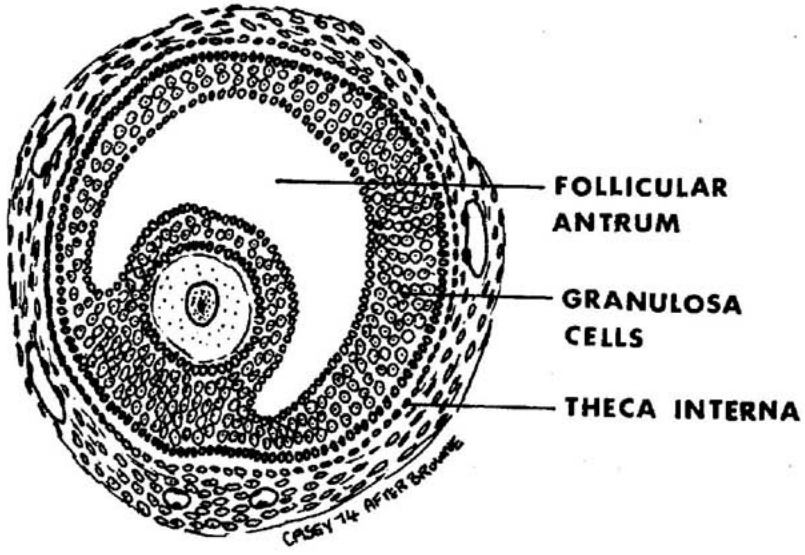

Fig. 16.1. Graafian follicle nearing maturity.

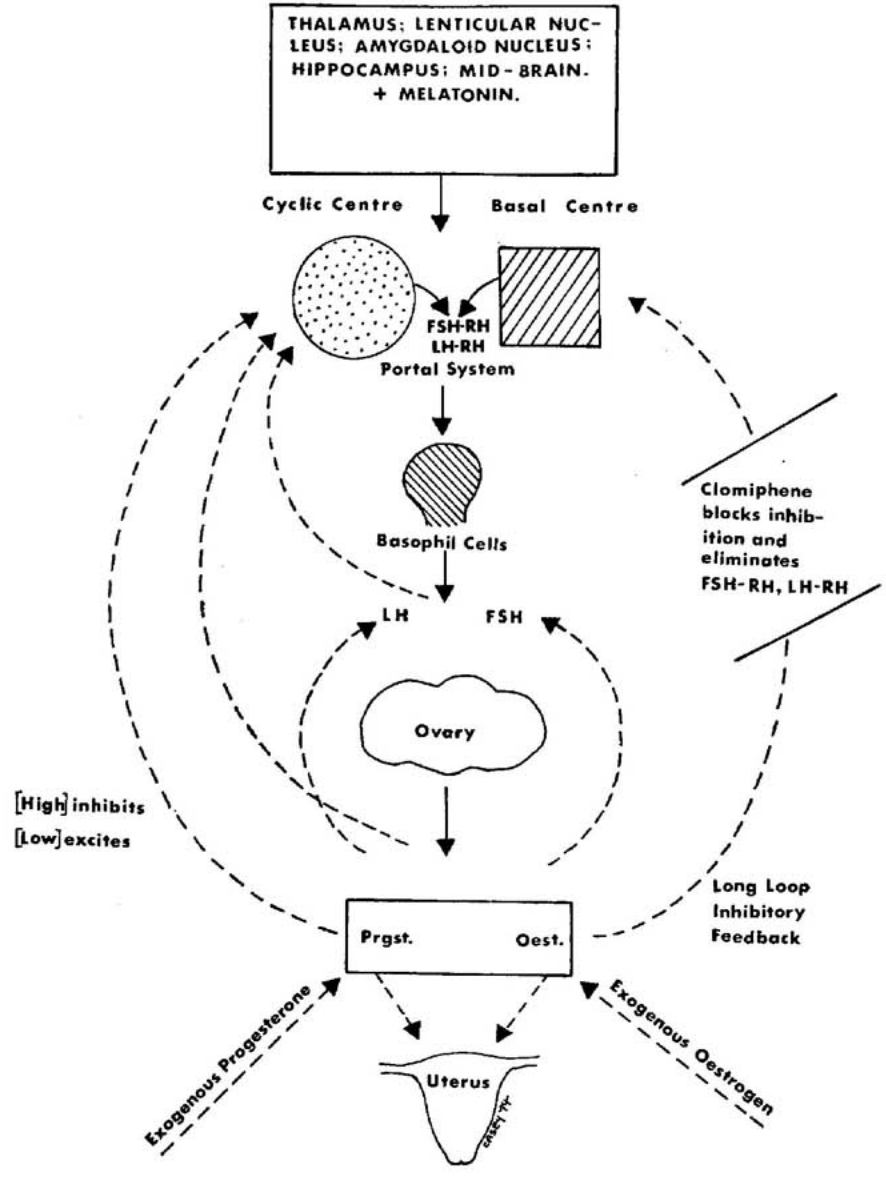

Fig. 16.2. Hormonal mechanisms in the reproductive pathway.

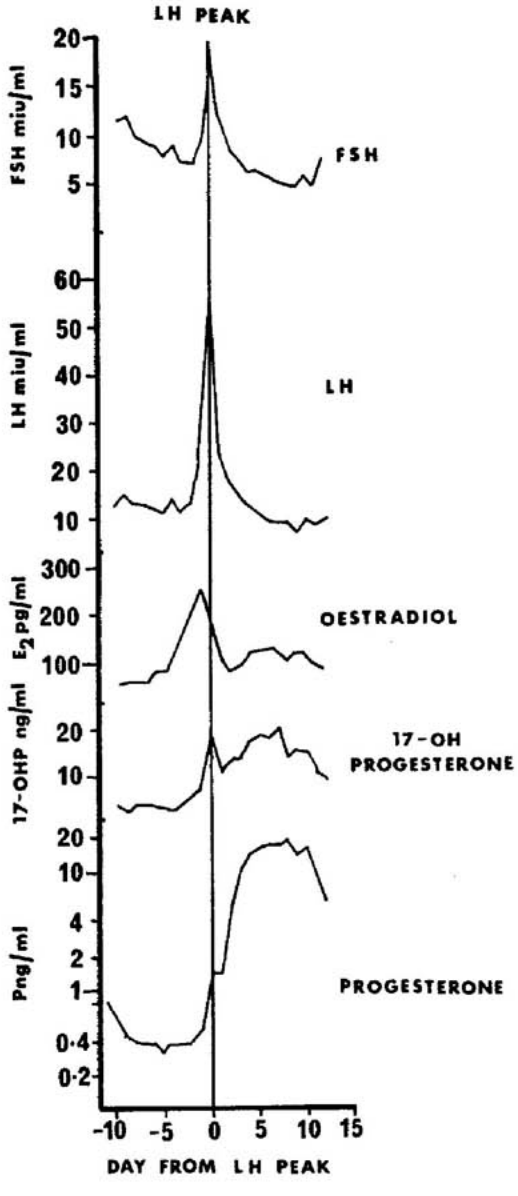

Fig. 16.3. Plasma hormone levels during the normal menstrual cycle.

Ovary.

a. *Oestrogen* in the ovary is produced by the granulosa cells and the theca interna (Fig. 16.1). It is also produced by the theca interna cells of the corpus luteum (yellow body – due to carotene). Oestrogen is carried in the circulation bound to B-globulins, albumin, and loosely to red blood cells. Excretion takes place via the gut and the kidneys (Fig. 16.4).

b. *Progesterone* is present in the peripheral blood in detectable amounts only after ovulation (Fig. 16.3). It is produced by the granulosa lutein cells of the corpus luteum. In the blood 95% of progesterone is bound to plasma proteins, mainly albumin. It is metabolized to pregnanediol, the major part of which is excreted in the bile conjugated with glucuronic acid. About 10% of pregnanediol appears in the urine.

Hormonal Mechanisms In Ovulation

In the sexually mature female secretion of F.S.H. at the beginning of the cycle stimulates the growth of the Graafian Follicle. As the follicle enlarges, secretion of L.H. stimulates oestradiol secretion. Oestradiol

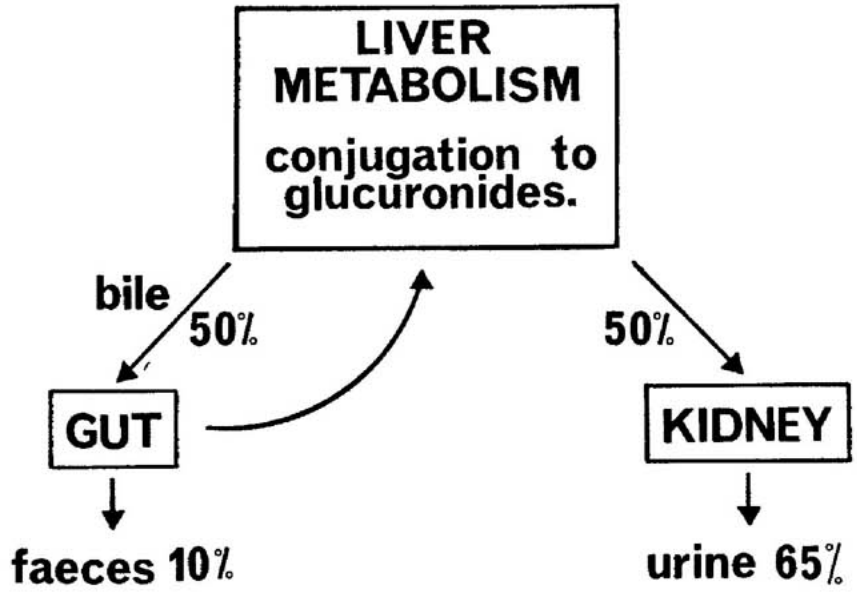

Fig. 16.4. Pathways of oestrogen excretion.

decreases the activity of the basal centre through the long feed-back loop, lowering the releasing hormone secretion. Because the half-life of L.H. is ten to fifteen times *shorter* than that of F.S.H. L.H., levels fall rapidly whereas F.S.H. levels only slowly. With the fall of the plasma L.H. ovarian oestradiol secretion decreases thus disinhibiting the basal centre which can produce more releasing hormone causing a significant output of both F.S.H. and L.H. The F.S.H. stimulates the growth of the follicle while the L.H. stimulates oestradiol secretion which is now at a higher level of production because of the greater manufacturing capacity of a large follicle. Such successive steps produce a peak in the plasma oestradiol concentration at about day 13 in a normal 28 day cycle (Fig. 16.2).

The oestradiol peak stimulates the cyclic centre in the hypothalamus, producing releasing hormone which in turn results in a large, mid-cycle L.H. peak.

If the Graafian Follicle is 0.8-1.2 cm. in size and has been present in the ovary for less than 54 hours, rupture of the follicle will take place. If the above conditions have not been fulfilled or the L.H. peak is small, leutinization of an unruptured follicle will take place.

A number of follicles enlarge in any one cycle, but usually the largest is ruptured while the others undergo atrophy. Mechanisms for rupture could include increase in the tension of *liquor folliculi*, contraction of ovarian unstriped muscle, degeneration of the capsule, or enzymatic action.

As the corpus luteum forms in the ovary, a small amount of progesterone is secreted (Fig. 16.3) This low concentration stimulates further L.H. secretion (Fig. 16.2 – long loop feed-back).Once higher levels of progesterone are secreted L.H. is depressed (Fig. 16.2). During the week after ovulation the falling levels of L.H act as luteotrophic hormone, stimulating progesterone production. The progesterone reaches its peak 5-8 days after ovulation and then falls again in the absence of either placental H.C.G. or pituitary L.H.

Following ovulation the oestradiol blood levels fall quite steeply and then rise more slowly as the corpus luteum develops. The second peak of oestrogen production (Fig. 16.3) corresponds to the maximum corpus luteum activity. The high oestrogen concentration inhibits the basal centre, lowering gonadotrophin levels (Fig. 16.2).

Conception and Implantation

At ovulation the abdominal ostium of the Fallopian tube and its sur-

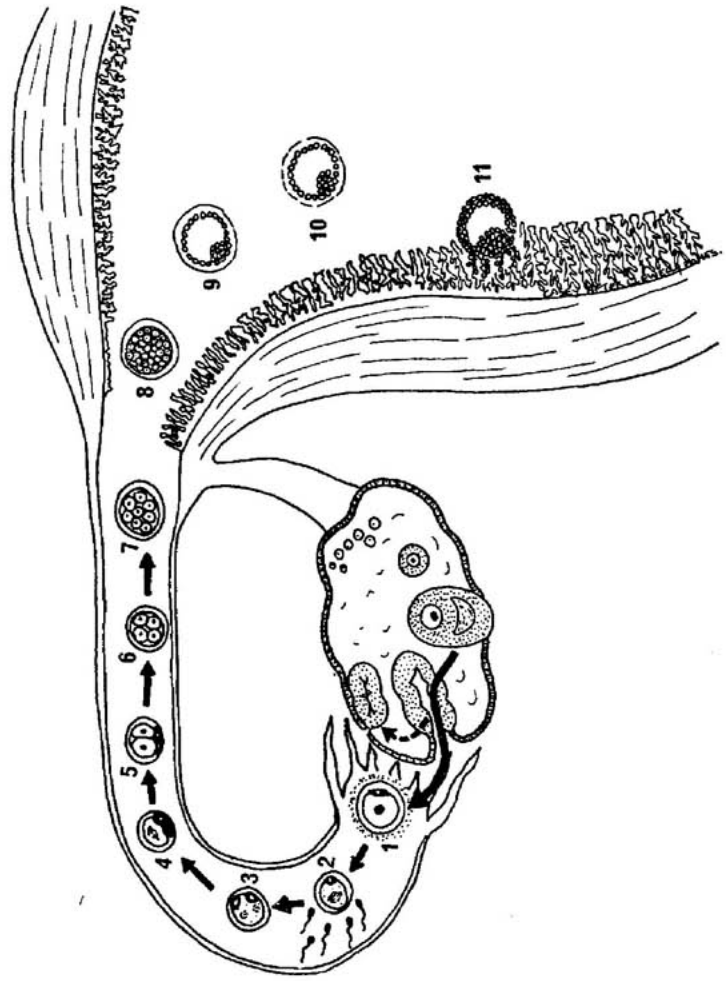

Fig. 16.5. The development of the ovum and its passage through the Fallopian tube into the uterine cavity.

1. Unsegmented oocyte;
2. Fertilization;
3. Pronuclei formed;
4. First spindle division;
5. Two cell stage;
6. Four cell stage;
7. Eight cell stage;
8. Morula;
9. and 10. Blastocyst formation;
11. Zona pellucida lost, implantation occurs.

rounding fimbriae spread over the ovary. At the same time the ovary *moves* so as to bring its surface into contact with the infundibulum of the tube. The result is that the ovum, surrounded by its granulosa cells (corona radiata) enters the tube rather than the peritoneal cavity (Fig. 16.5). Peristaltic movements in the tube, greatest about the time of ovulation (Woodruff *et al*, 1969), propel the ovum towards the uterine cavity. Unfertilized, the ovum can survive about 18 hours in the tube.

In the ampulla during fertilization, the ovum is surrounded by spermatozoa one of which penetrates and deposits its pronucleus within the ovum (Fig. 16.5). By this time the second maturation division has taken place in the oocyte, forming the second polar body. The female and male pronuclear material form chromosomes which fuse forming the zygote (Fig. 16.5). Cell division now takes place the ovum passing through increasingly numerous cell stages.

The ovum travels 5-7 days in the tube before implanting. As cell division reaches the morula (mulberry) stage and the dividing ovum settles between the folds of the secretary endometrium, fluid from the uterine glands enters the cell mass where it collects to form a central cavity. The ovum has now reached the blastocyst stage and measures some 0.13 mm. in diameter.

The zona pellucida breaks down at this stage (Fig. 16.5). The blastocyst touches the endometrium and becomes attached. Adherence is followed by invasion, possibly through alkaline disintegration of the underlying epithelium, while primitive trophoblast cells form all over the surface of the blastocyst. By day 8 implantation has begun and 3 to 4 days later it is complete. Some vaginal bleeding may occur when a blood vessel is eroded. This is the placental sign or implantation bleed and may be mistaken for a light period.

B. Infertility and Subfertility

Definition. Subfertility is defined as failure to conceive after a year of normal coitus. Seventy percent of women desiring to become pregnant do so within 12 months and 85% within two years. A subfertile patient who has never conceived is suffering from primary subfertility. Secondary subfertility is applied to a patient who has difficulty conceiving after a previous pregnancy.

Infertility is the absolute absence of the ability to conceive.

Incidence. About 10% of married couples fail to achieve a pregnancy between puberty and menopause.

Aetiology. There are many factors capable of upsetting the normal reproductive pathway. These are presented in a summarised way in Table 16.1. One should keep in mind that more than one barrier to reproduction may be present in a couple presenting for investigation.

Male factor	25%
Gross pelvic pathology (female)	12%
Cervical factor	10%
Uterine factor	4%
Tubal factor	50%
Ovarian factor	4%
Psychosomatic factor	30%

Note: Many couples have multiple factors operating.

Table 16.1

Factors influencing fertility in 1450 couples (Llewellyn-Jones, 1973).

The following account is an elaboration of the factors from Table 16.1.

1. The Male Factor:

Oligospermia or Azoospermia. These terms refer to reduced number or absence of spermatozoa respectively. Sperm may also be abnormal. On *semenalysis* the volume, count, motility, and morphology of sperm are assessed (Table 16.2). Because of the great variation between counts at least *three* are needed to reach any conclusion.

Volume	2ml. (normal average 2.5 - 4 ml)
Count	20,000,000 per ml. (normal average 60,000,000 per ml)
Motility	more than 40% after 4 hours.
Normal forms	more than 60%

Table 16.2

Lower limits of normality on semenalysis.

Causes of Oligospermia and Azoospermia:

1. Testicular atrophy – e.g. mumps orchitis.
2. Varicocele – raised temperature reduces sperm count.
3. Clothing – tight underpants raising testicular temperature.
4. Undescended testis – abnormal testis or raised intra-abdominal temperature.
5. Kleinfelter's syndrome – small testes, gynecomastia, infertility.
6. Epididymal block – previous inflammatory disease; herniorraphy.
7. Fear, anxiety, fatigue – can reduce sperm count.

Other Male Factors

Impotence (failure of erection)–psychological causes, diabetes (Rubin 1958), nerve lesions.

Hypospadias – severe degrees prevent sperm deposition. Premature ejaculation.

2. **Cervical Factors:**

1. "Chronic cervictis" (cervical erosion) and endocervicitis with purulent cervical mucus may interfere with passage of sperm.
2. Vaginal infection – e.g. trichomonas vaginalis.
3. Hostility of cervical mucus – Mucus may be too thick for penetration by sperm. This may be improved by a three day course of oestrogen, e.g. stilboestrol 0.5 mg just before ovulation, which will not be suppressed by this dose.
4. Anti-Semen antibodies. This is a rare cause.

3. **Uterine Factors:**

1. Congenital abnormalities – usually can conceive but may abort.

2. Pelvic T.B. – tuberculous endometritis may preclude implantation.
3. Fibromyomata – may interfere with implantation or cause abortion.
4. Intrauterine adhesions – (Asherman's syndrome) cause repeated early abortions rather than infertility.
5. Retroverted uterus – infertility can occasionally be rectified by correction of retroversion.

4. **Tubal Factors:**

1. Tubal spasm – associated with anxiety and tension. It may also be the result of instrumentation during hysterosalpingography. These are functional occlusions.
2. Fimbrial adhesions – from previous pelvic peritonitis or endometritis.
3. Hydrosalpinx – fimbrial block due to previous salpingitis filling with secretions.
4. Mid-tubal block – inflammatory aetiology.
5. Cornual block – inflammatory aetiology.
6. Previous salpingectomy or ligation.

5. **Combinationed Factors:**

Some 3% of couples seeking advice on infertility have not consummated their marriage. Ignorance as to when (during the cycle) to have intercourse is an important factor.

6. **Ovarian Factors:**

In this section a discussion of various causes of ovulatory failure are in order. However, to save repetition the entire aetiology of *amenorrhoae* will be included here.

Amenorrhoea may be defined as the absence of menstruation for a period which is twice that of the normal menstrual cycle of a woman who has menstruated previously (secondary amenorrhoea); or the

non-appearance of menstruation in a girl who has reached the age of 16 (primary amenorrhoea).

a. *Physiological Amenorrhoea*

i. *Before Menarche* – The hypothalamus has not yet initiated cyclic hormonal release.

ii. *Pregnancy* – Amenorrhoea persists due to increasing levels of oestrogen and progesterone.

iii. *Lactation* – Ovulation is suppressed due to prolactin inhibition of gonadotrophin production.

iv. *Postmenopausal* – The ovary fails to respond to gonadotrophins.

b. *Pathological Amenorrhoea*

i. *General* – Acute and chronic infection, e.g. hepatitis and T.B.
- Malignancy, malnutrition, anorexia nervosa, and uncontrolled diabetes.

ii. *Local*

1. Cryptomenorrhoea – Imperforate hymen
   - Vaginal atresia
   - Cervical stenosis
2. Surgical intervention – Oophorectomy, hysterectomy, over irradiation
3. Hermaphroditism – Both testes and ovaries coexist
4. Pseudo-hermaphroditism – female genitalia exist with male gonads.
5. Gonadal dysgenesis – primordial germ cells fail to reach ovary during developmental stage – failure of ovarian function.
6. Congenital absence of the uterus due to failure of maturation of Mullerian tissue.
7. Pelvic disease such as tuberculosis

iii. *Genetic*

Turners Syndrome – XO genotype with dwarfism and webbing of neck.

iv. *Endocrine*

1. *Cerebral* – Emotional stress. A common cause.
2. *Hypothalamic* – Suppression of releasing factors by various forms of the "pill", reserpine, phenothiazines, etc.
   - Hypothalamic lesion, e.g. Chiari–Frommel syndrome. This is an association of post-partum galactorrhoea, super-involution of the uterus, and amenorrhoea.
   - Frohlich's syndrome (Dystrophia adiposogenitalis) May be a primary hypothalamic disturbance which, operating from childhood results in genital hypoplasia, amenorrhoea, obesity, and somnolence.
3. *Pituitary* – Acromegaly (Eosinophilic adenoma).
   - Simmond's disease, including Sheehan's syndrome, which is post-partum ischaemic necrosis of the pituitary due to haemorrhage and shock of childbirth.
4. *Ovarian* – Metropathia Haemorrhagica (Schroeder's disease). Amenorrhoea followed by menorrhagia (see page 369).
   - Stein-Leventhal Syndrome – Amenorrhoea or oligomenorrhoea, obesity, hirsuties, infertility, and enlarged (polycystic) ovaries. There is a 19-hydroxylase deficiency in the ovary leading to defective oestrogen production and rechanelling of steroids into androgen production.
   - Arrhenoblastoma of ovary. This is an androgen-producing tumour which inhibits the hypothalamic cyclic centre.
   - Oestrogen-producing tumours (Granulosa cell tumour)

5. *Adrenal* – Adrenogenital syndrome (Congenital adrenal hyperplasia) – A condition caused by a group of enzyme defects; (21 -hydroxylase in 90%, 11-B-hydroxylase in 10%) which prevents cortisone synthesis from progesterone thus raising ACTH levels. Adrenal androgen production increases as a result, causing masculinization and inhibition of the cyclic centre.
   – Adrenal tumour producing androgens.

6. *Thyroid* – Hyperthyroidism may result in amenorrhoea. Hypothyroidism may result in menorrhagia.

b. *Management of Infertility*

When dealing with the problem of infertility it is important to remember that one is investigating an infertile *couple* and not the woman alone. Once investigations are completed the results should be discussed with the couple. A completely hopeless prognosis should never be given, because women with seriously diseased pelvis have conceived.

Procedure:

. Full histories of both husband and wife are recorded.
. Full general physical examinations are performed, including a pelvic examination of the wife.
. Investigations – Post coital test
   – Endometrial biopsy
   – Hysterosalpingogram
   – Basal body temperature chart for at least three months.

These investigations are basic, and even though an abnormal factor is found during the investigation, it is mandatory that they be performed before undertaking treatment. There is little point in submitting a woman to surgery in an attempt to relieve tubal obstruction if her husband is azoospermic.

Investigations:

1. **Male Factors**

a. *Post-coital Test* (Sim's test; Sim's -Heubner test)
Four hours after intercourse on day 14 of the cycle the wife presents herself at the clinic. Mucus is removed from the cervical canal for immediate microscopy. The presence of at least 2 to 3 motile and progressing sperms per high power field indicates a positive result and needs no semenalysis.
If on post-coital test there are:

i. reduced number of sperm;
ii. no sperm;
iii. or dead sperm,

semenalysis is carried out (see page 16.4), and infertility due to male factor is excluded.

. *No sperm* will be seen with:

a. Azoospermia, e.g. Kleinfelter's syndrome
b. Failure to ejaculate, premature ejaculation, and hypospadias
c. Failure to reach the cervix, e.g. apposition of vaginal walls due to obesity. The problem may be solved by a posterior position during intercourse.
d. Epididymal block. Reconstructive surgery is about 15% successful.

. *Reduced sperm* count may be seen with:

a. Tired husband (Prescribe a holiday!)
b. Varicocele, tight underclothing, testicular atrophy. Surgery for varicocele is 15% successful with respect to sperm count.

. *Dead sperm* may be seen if there is:

a. Male genitalia area infected killing the sperm
b. Local infection in the female producing sperm toxicity
c. Antibodies to sperm in the female partner. Difficult to treat. Abstinence or the wearing of a condom for 6 to 9 months may reduce antibody production. Cautery of cervical glands may help.
d. Sperm not maturing properly.

b. *Cervical Mucus Invasion Test*

In this test attraction and penetration of the cervical mucus by the sperm can be visualized under the microscope. Mid-cycle cervical mucus and a masturbation specimen of sperm are placed on a slide with the cover slip applied. If sperm is initially alive it will enter normal mucus as an invading column.

Infection will cause sperm to move away from the mucus. Presence of antibodies may retard and kill the sperm.

2. **Tubal Factors:**

In the history look for evidence of gonorrhoea, puerperal infection, endometriosis, and peritoneal infection.

a. *Tubal Patency Tests*
   i. *Rubin's Test.* Tubal insufflation with CO_2 (carbon dioxide rapidly dissolves in blood and is less likely to cause embolism) is usually done on an outpatient basis. A pressure tracing is recorded on a kymograph. A low-pressure tracing (Fig. 16.6), the sound of gas passing through the tubes on auscultation, and shoulder pain with subdiaphragmatic irritation on sitting up indicate patency of at least one tube. A negative result could indicate tubal spasm and should be repeated after one month or confirmed by hysterosalpingography. Contraindications to insufflation include uterine haemorrhage, pelvic infection, and purulent vaginal discharge.
   ii. *Hysterosalpingogram.* This is a radiological examination after intrauterine injection of radio-opaque medium. It is more informative than a Rubin's test as it may reveal uterine abnormalities or tubal pathology. The site of obstruction may be seen (with tubal spasm no tube outline will be seen at all). To test for adhesions a second film is made 20 minutes after the first to look for spread of the dye. Hysterosalpingography is done before ovulation is expected so as not to interfere with a possible pregnancy. Contraindications are the same as for insufflation.

b. *Laparoscopy.* With the abdominal cavity inflated with gas a laparoscope is introduced through an subumbilical incision and the fimbrial ends of the Fallopian tubes are identified. Methylene blue dye is injected through the cervix into the uterus and peritoneal spill noted. The general morphology and possible pathology of pelvic structures is observed.
Treatment. Fimbrial adhesions, hydrosalpinx, tubal block and previous tubal ligation all require specialised microsurgical treatment. Success rate varies between 15% and 35% for these procedures.

3. **Ovarian and Endocrine Factors**

Positive proof of ovulation is either a pregnancy or laparoscopic aspiration of a ruptured Graffian follicle. Ovulation is *presumed likely* if on history there is:

a. A regular 28 day cycle,
b. Mucous discharge at midcycle,
c. Progesterone effects:
   – regular cycle,
   – pre-menstrual (spasmodic) dysmenorrhoea,
   – breast effects (tender and full premenstrually).

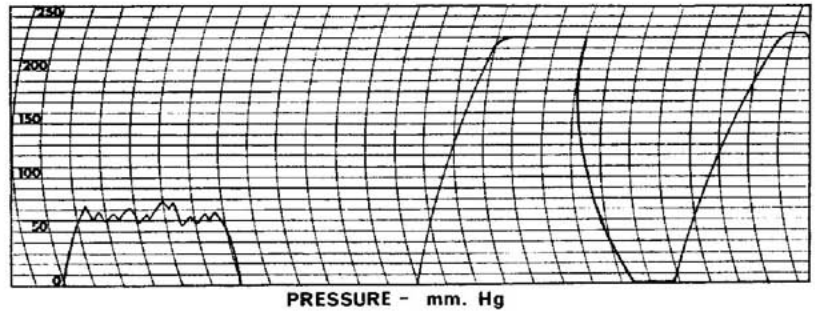

Fig. 16.6. Tracings of carbogen insufflation of the tubes.
1. The pressure rose to 70 mm. mercury after which the gas passed into the peritoneal cavity.
2. In this case gas failed to pass dispite a pressure of 230 mm. mercury.

Evidence of ovulation may be presumed if:

1. Secretary change is seen premenstrually on *endometrial biopsy* indicating progesterone effects from the corpus luteum.
2. Biphasic *basal body temperature* chart (Fig. 16.7). Following ovulation the temperature rises 0.2 to 0.6°C.
3. Change in the *cornification index*. Oestrogen causes an increase in the number of squames in smears from the lateral vaginal walls. Progesterone causes a reduction in the number of squames and an increase in the number of intermediate sized cells with a reticulated nucleus. The cornification index therefore normally falls after ovulation. Clumping and turning up of cell edges as well as a leucocytosis are also present.
4. *Cervical mucus* at ovulation can be drawn out into long strands and on drying shows "ferning" arrangement of sodium chloride crystals. After ovulation cervical mucus is thicker and the "ferning" pattern will not form if progesterone is present.

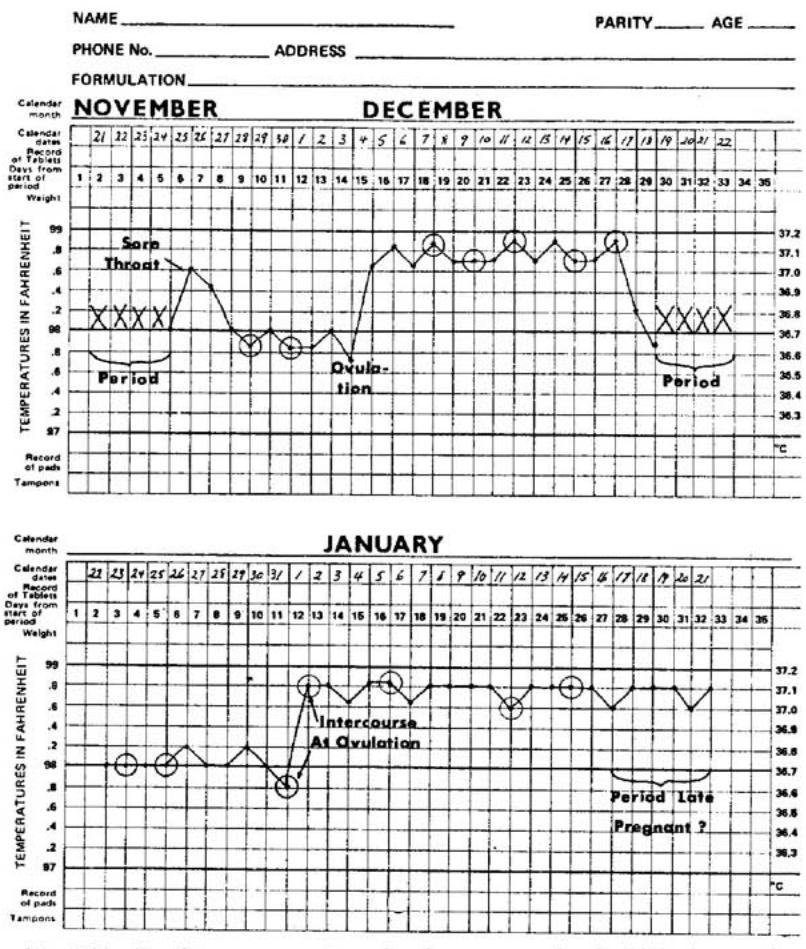

Fig. 16.7. Basal temperature chart showing an example of a biphasic record, and an ovulatory record.

5. Levels of *plasma progesterone* of over 5ng/ml constitute evidence of ovulation. A leutinized unruptured follicle may produce levels of up to 3ng/ml.

If there is an *absence of ovulation* the level at which the ovulatory mechanism is blocked may be determined.

1. *Primary ovarian failure* is associated with high levels of F.S.H. and L.H. (as in menopause) and is usually accompanied by hot flushes. Ovarian capacity may be tested directly by giving injections of pituitary gonadotrophins and measuring plasma oestradiol. No treatment is available for primary ovarian failure. Adoption should be considered.

2. *Pituitary failure* is reflected by low gonadotrophin levels and absence of hot flushes, and may be tested by intravenous injections of gonadotrophin releasing hormone to produce an L.H. peak within 30 to 40 minutes.

3. *Combined hypothalamo-pituitary failure* is the most common of these and the test for function is the same as the treatment.

Treatment:

Patients suffering from ovulatory failure due to psychogenic causes, primary amenorrhoea with normal or low gonadotrophins, Stein-Leventhal syndrome, and post-pill infertility, may benefit from clomiphene citrate (Clomid) by mouth, or Human Pituitary Gonadotrophin by injection.

Since clomiphene citrate is less expensive and simpler to use it is usually tried first. H.P.G. is reserved for those cases which have not responded to clomiphene citrate. Clomiphene citrate in humans probably acts by blocking the long loop inhibitory effect of oestrogen on the hypothalamic "basal centre" (Fig. 16.2). It is thus an "antioestrogen". The result is the secretion of gonadotrophin releasing factors by the hypothalamus, which acts on the pituitary to secrete F.S.H. and L.H. The gonadotrophins stimulate follicle maturation and oestrogen production, which triggers an L.H. peak bringing about ovulation.

In the Stein-Leventhal syndrome a similar effect may be achieved by a resection of a wedge of ovary. This will reduce the amount of androgen produced disinhibiting the cyclic centre. Clomiphene citrate is given initially in a dose of 50mg twice daily, for five days. Side effects may include abdominal discomfort due to enlargement of follicular or lutein cysts, hot flushes from gonadotrophin production, blurring of vision, and hair loss. Mulitple pregnancy may result in 10% of cases.

Human Pituitary Gonadotrophin is administered as a daily injection for 8 to 9 days. The aim is to bring no more than one Graffian follicle to maturity, and then to rupture the follicle with an artificial L.H., F.S.H. peak after which fertilization can take place. If the response to H.P.G. is excessive, urinary oestrogens rise too rapidly indicating multiple follicles. Excellent control must be maintained to produce a single foetus.

Other investigations for Amenorrhoea may include:

. Chromosomal studies.
. Total 17-ketosteroids.
. Thyroid function tests.
. Pituitary fossa X-rays.

D. Contraception

Control of fertility is an ever increasing problem in the world because of the general fear of overpopulation, the awareness of the necessity to control family size for financial, health, as well as other considerations, and the increasing sophistication of modern society demanding either instant contraception or instant fertility.

There are numerous methods of contraception. The choice of the correct method will be more easily made if the following axioms are kept in mind.

. Any method of contraception is better than no method.
. The most effective method is one that the couple will use with the greatest consistency.
. Acceptability is the most critical factor in the use and the effectiveness of a contraceptive method.

Methods of contraception may be subdivided into those used by:

1. Both partners;
2. The male, and;
3. The female.

1. Methods used by both partners:

a. *Continence.*

b. *Rhythm Method.* This means avoidance of coitus around the time of ovulation. In a woman with a regular 28 day cycle ovulation takes place about the 14th day. Allowing for slight irregularities and sperm survival, intercourse may *NOT:* take place from day 9 to day 19 of the cycle–or to be safer still, from day 7 to day 21.

Advantages – Acceptable to the Roman Catholic Church.
Relatively simple procedure.

Disadvantages

– Cycles must be regular.
– Requires an intelligent patient,
Requires exercise of continence over a period of time.
– High failure rate. (Table 16.3)

Contraception – Failure Figures

Method		Pregnancy per 100 woman years
Female sterilization		0.02
Vasectomy		0.02
Oral contraceptives	Combined	0.5
	Reverse sequential	0.7
	Incremental	1.0
	Sequential	2.0
IUCD — copper		2.0
IUCD		3.0-5.0
Cervical cap		8.0
Condom		10
Diaphragm		15
Rhythm method		20
Spermicides e.t.c.		20
Coitus interruptus		25

Table 16.3

2. Methods used by the Male:

a. *Condom* (Sheath; Protective; French letter)

Advantages
– Simple to use.
– Male's responsibility.
– Mechanical barrier to infection.

Disadvantages
– Expensive,
– Diminished sensation,
– Lack of spontaneity,
– High failure rate – mostly due to faulty techinique. Defect rate is 0.25 to 0.89.
– Danger of rupture 1/150-300.

b. *Coitus Interruptus* ("withdrawing"). This is probably the most common method used. Related methods are coitus interfemora, and coitus reservatus.

Advantages – Simple and easy to understand.

Disadvantages – Much self control needed by both partners.
– Often unsatisfying.
– High pregnancy rate (Table 16.3).

c. *"Vasectomy"* – Surgical removal of a section of the vas deferens, the remaining ends being turned back on themselves. The patient should be warned that sterility is not effective for some *three months*, in which three seminal analyses (see page 333.) one month apart must show complete absence of sperm.

Advantages – Simple technique – can be done on "out-patient" basis under local anaesthesia.
– Little morbidity.
– Very effective.

Disadvantages – Surgery is necessary.
– Male (and female) attitudes towards the procedure often prevent its acceptance.
– Not totally reversible (only in 10-40% of cases). Reversibility must not be a consideration therefore. Consider also the death of children, or a divorce.
– Long post-vasectomy time before "safe" period is reached.
– Theoretical possibility of late "auto-immune" effect to one's own sperm.

3. **Methods used by the Female:**

a. *The Diaphragm* (Dutch cap) – Fig. 16.8.
Once inserted the diaphragm must be left in place for at least 6 hours after intercourse and no longer than 16 hours, as the rubber and prolonged retention can cause an unpleasant discharge. The safest technique is to insert the diaphragm nightly. A spermicide should be used as well.

Advantages – Relatively simple to use,
– Woman's responsbility,
– Inexpensive.

Disadvantages – Doctor is required to instruct the patient and to fit the device.
– Must be used regularly.
– Anatomical variations may make fitting impossible.
– Psychological fear of touching the genitalia and general patient unwillingness may prevent use.
– Lack of spontaneity unless fitted nightly.

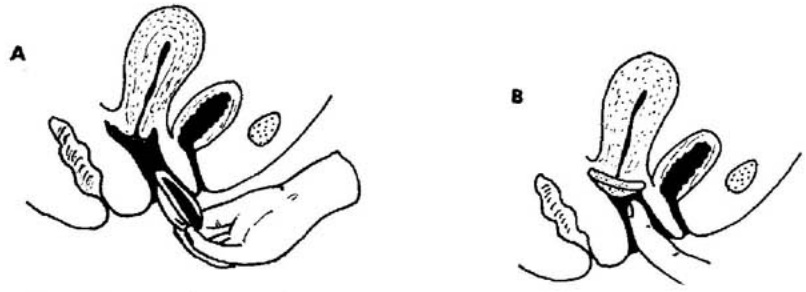

Fig. 16.8. Insertion of a diaphragm.
A. The diaphragm is smeared with spermicidal cream round the edges and on both sides, and guided into the posterior fornix.
B. The front end is tucked up behind the symphysis.

b. *Cervical Cap.* A plastic or metal cap fitting over the cervix and left in place for days. A spermicide must be used.

Advantages – May be left in place for days,
– Cheap and will last,
– Womans responsibility.

Disadvantages – Doctor required to instruct patient and to fit the device.
– Not effective for abnormal cervices.
– Technique difficult to learn.
– Psychological fear of touching the genitalia and general patient unwillingness may prevent use.

c. *Douches* (Post-coital). Water or chemical douching is very unreliable, and often induces pathological infestation of vagina by washing out the normal vaginal flora.

d. *Chemical* – types
1. Gels, creams, and pressure pack vaginal foams,
2. Vaginal foam tablets,
3. Vaginal pessaries,
4. Sponge and foam,
5. Sponges and tampons with household spermicides.

Advantages
- Simple to use.
- Most are inexpensive.

Disadvantages
- Often messy.
- Equipment must be at hand. Some require much preparation.
- A few are not effective for a time after insertion.
- High pregnancy rate.

e. *Intra-Uterine Contraceptive Devices* (I.U.C.D.) (Fig. 16.9 and 10.) This is an age-old technique. The Arabs, for many centuries

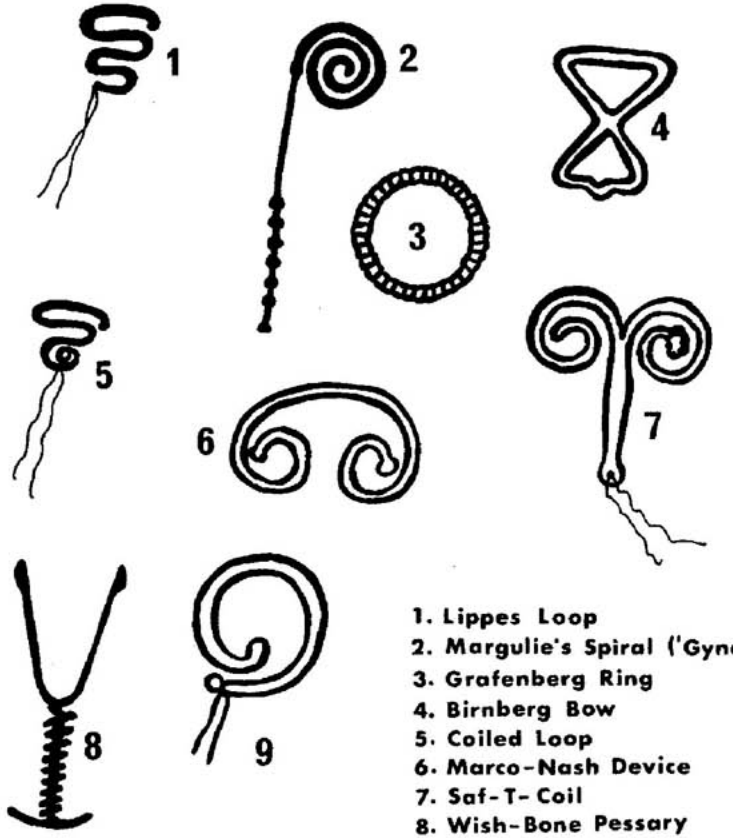

Fig. 16.9. The various models of intra-uterine devices. I.

past have used small intra-uterine stones to prevent pregnancy in camels. Experience in this century dates back to Grafenberg's first paper in 1928 (a 10 year series). He used rings of silver and gold wire.

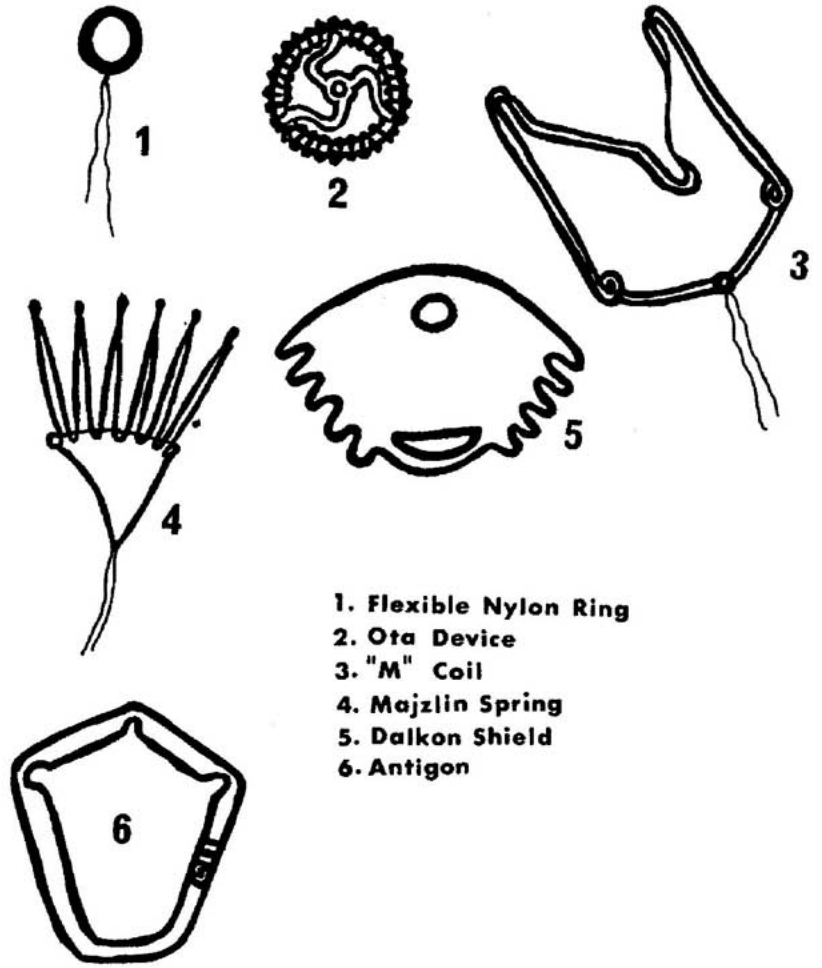

Fig. 16.10. The various models of intra-uterine devices. II.

The IUCD's did not gain popularity until the 1960's when plastic models with the common properties of flexibility,

shape retention, ease of introduction, and non-irritability to the uterus, were produced.

Mode of action: The mode of action of the IUCD's is uncertain. They do not cause obstruction to sperm ascent, hormone imbalance, or endometritis. Three theories are propounded:

i. The IUCD accelerates tubal peristalsis hindering implantation.
ii. The encroachment of the IUCD on the endometrium acts as a physical barrier to implantation.
iii. The IUCD may cause a pre-clinical abortion.

The inert plastic IUCD's do *not* cause cancer, decreased fertility, ectopic gestation or foetal injury (if accidental pregnancy occurs).

At present the types of IUCD used at the Royal Hospital for Women, Paddington, are the Lippes Loop, and the Dalkon Shield.

Advantages
- Usually easily inserted.
- Acts immediately after insertion.
- Complete return to normal after removal.
- Does not irritate either partner.
- Very good in those patients with little motivation.
- Effective contraceptive, (Table 16.3).

Disadvantages
- Must be inserted by a doctor or trained technician.
- Expulsion, (10% with Lippes Loop).
- Uterine perforation, (1/2500 with Lippes Loop).
- Reactivation of pelvic infection. (1% with Lippes Loop).
- Side effects (40% with Lippes Loop).
  a. Bleeding – usual after insertion.
  b. Pain – "abdominal cramps".
  c. Discharge – Commonly increased normal discharge.

f. *Intra-Uterine Contraceptive Device plus Copper*

Metallic copper is thought to greatly enhance the biological effect of the IUCD (Fig. 16.11). These are still under trial, but the "COPPER 7" promises to be an excellent device when it is finally available for general use.

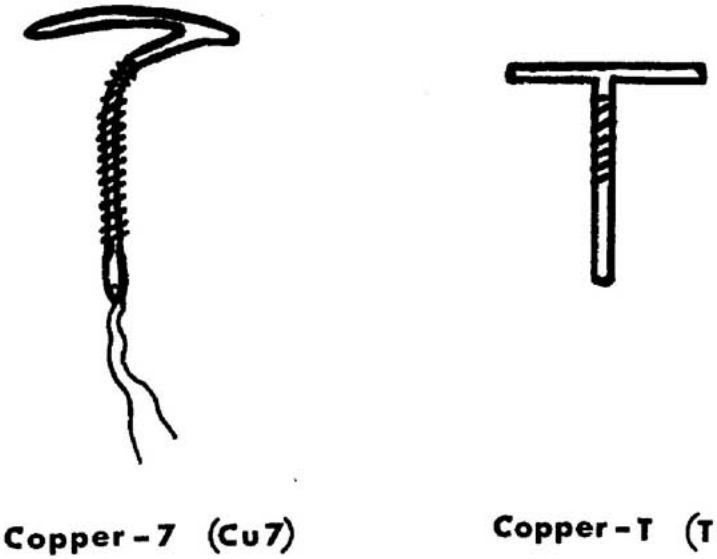

Fig. 16.11. Intra-uterine contraceptive devices with added copper.

g. *Surgical Sterilization*

1. *Tubal laparoscopic cautery:*

The Fallopian tubes are catuerized in the isthmic region using a laparoscope.

Advantages
- Relatively simple.
- Little disturbance to the patient as compared with the formal tubal ligation.
- As effective as some oral contraceptives (sequential).

Disadvantages
- Requires a general anaesthetic,
- Peritoneal cavity is entered,
- Possibility of damage to other organs,
- Not as effective as ligation.

2. *Tubal Ligation, Partial Excision, or Excision*

Advantages
- Relatively simple.
- Effective (Table 16.3).

Disadvantages
- Requires a general or regional anesthetic.

- The peritoneal cavity is opened – abdominally or vaginally.
- There is an associated morbidity and mortality.

h. *Hormonal Contraception*

Ovulation may be suppressed either with oestrogens or progestogens. These act at the level of the hypothalamic centres (Fig. 16.1) suppressing the output of F.S.H. and L.H. releasing factors.

Types of Oral Contraceptives

In commercially available preparations in Australia the two oestrogens used are *ethinyl oestradiol* and its 3-methyl-ether, *mestranol* (Table 16.4). On a weight for weight basis ethinyl oestradiol is nearly twice as active as mestranol (Delforage *et al*, 1970) and has a slightly better therapeutic to toxic ratio than mestranol. In order to produce consistent ovulatory suppression ethinyl oestradiol in a dose of 0.05 mg daily has to be given for seven to fourteen days prior to ovulation (Carey *et al*, 1972). Effectiveness of suppression of ovulation is highest when tablets are begun on the first day of the cycle (beginning of menstruation). Oestrogens used alone however, may produce irregular, prolonged, and unpredictable menstrual bleeding patterns. For this reason it is necessary to use a progestogen with the oestrogen.

Synthetic steroids capable of producing a secretary change in the endometrium are known as progestogens. A progestogen alone in a dose equivalent to 0.4 mg norethisterone, will suppress ovulation if given for a week prior to the expected date of ovulation. It is not commonly used however, because irregular bleeding, polymenorrhoea and amenorrhoea will occur in 25% of patients. When the progestogen is used for more than 7 days there is an increased frequency of breakthrough bleeding. To overcome these problems an oestrogen must be combined with the progestogen.

The "combined" type of oral contraceptive is the most common one on the Australian market (Fig. 16.12) and includes all brands apart from the sequential and serial preparations of Ovin, Serial C and Serial 28. The "mini" pill and other types of preparations shown in Fig. 16.12 are not yet commercially available although these may be prepared by the practitioner from commercially available oestrogen and progestogen preparations, to suit the patient's particular requirements.

TABLE 16.4 COMMERCIALLY AVAILABLE CONTRACEPTIVES

		Oestrogen content (mg)			Progestogen content, (mg).								
	POTENCY –	1	1/2	5	10	1	1/10	3	1		1	0.5	
Product	Manufacturer	Ethinyl Oestradiol	Mestranol	d & l* Norgestrel	d Norgestrel	Norethisterone Acetate	Norethynodrel	Ethinodiol Diacetate	Lynestrol	Dimethisterone	Norethisterone	Megestrol Acetate	
Ovin	Mead Johnson	100								25			
Orgaluton	Organon		150						5.0				
Conovid	Searle		75				5.0						
Anovlar 21	Schering A.G.	50				4.0							
Volidan 21	A. & H.	50										4.0	
Gynovlar 21	Schering A.G.	50				3.0							
Oralyn 22	Adams		100				2.5						
Anacyclin	Ciba		75						2.5				
Lyndiol 2.5	Organon		75						2.5				
Norlestrin 28	Parke Davis	50				2.5							
Conovid E	Searle		100				2.5						
Norinyl 2	Syntex		100								2.0		
Ortho Novum 2	Ethnor		100								2.0		
Microcyclin	Ciba		100						1.0				
Ovostat	Organon		100						1.0				
Ovulen	Searle		100					1.0					
Ortho Novum 1/80	Ethnor		80								1.0		
Edulen	Searle	50						1.0					
Norinyl 1 & 28	Syntex		50								1.0		
Minovlar 21 & Ed	Schering A.G.	50				1.0							
Orlest 28	Parke Davis	50				1.0							
Ortho Novum 1/50	Ethnor		50								1.0		
Ovulen 1/50	Searle	50						1.0					
Ovulen 0.5	Searle		100					0.5					
Ovulen 5.50	Searle	50						0.5					
Eugynon 0.5 & Ed	Schering A.G.	50		0.5									
Ovral 21	Wyeth	50		0.5									
Ovral 28	Wyeth	50		0.5									
Serial C	A. & H.	100										1.0	
Serial 28	A. & H.	100										1.0	
Neogynon & Ed	Schering A.G.	50			.25								
Nordiol 21	Wyeth	50			.25								
Nordiol 28	Wyeth	50			.25								

*The 1 form of d1 Norgestrel is inactive.
Potencies of oestrogens are based on an arbitrary potency of *one* assigned to ethinyl *oestradiol.*

Potencies of the progestagens are based on an arbitrary potency of *one* assigned to *norethisterone.*

The combined pill depends on progestogen for suppression of ovulation, the oestrogen being added to prevent breakthrough bleeding and to

TYPES OF ORAL CONTRACEPTIVES

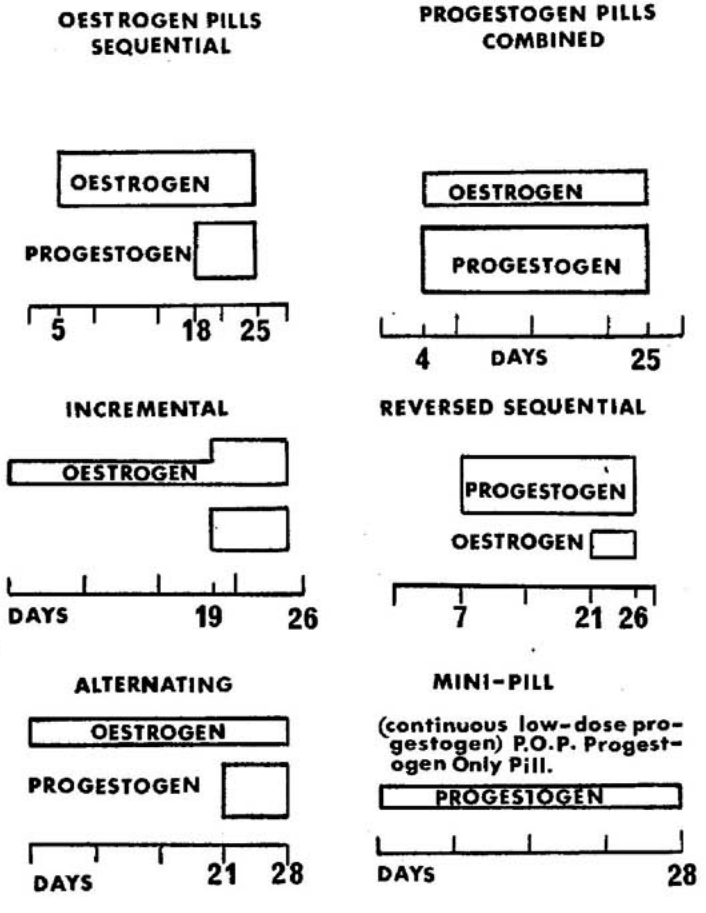

Fig. 16.12.

promote a predictable and satisfactory menstrual flow two days after finishing the hormonally active tablets. The numerous commercially available preparations vary in their content of oestrogens and progestogens (Table 16.4). Thus a patient sensitive to progestogen or oestrogen and suffering from their side effects may be placed on a preparation containing a lower amount of the particular hormone. It is important at this stage to realize that it is *not* simply the amount in weight but rather the *activity and the amount* of the hormone that needs to be taken into account when comparing the relative potency of the various available brands (Table 16.4). Thus OVULEN, for example, containing 1.0 mg of ethinodiol diacetate, carries three times the activity of 1.0 mg of norethisterone, but a 30 fold activity over that of 1.0 mg norethynodrel. The relative potencies of the various progestogens and oestrogens are given in Table 16.4. One ought to keep in mind the fact however, that lowering the doses of hormones must be weighed up against slightly reduced reliability. The most reliable oral contraceptives commercially available therefore, are the medium to high dose combined preparations, such as OVULEN or EDULEN.

The *sequential* oral contraceptive (Fig. 16.12) is made up of a sequence of oestrogen and combined oestrogen and progestogen tablets, commencing on the third or the fifth day of the cycle (depending on the preparation used). Because it is begun so late in the cycle a comparatively high dose (100 mg) of ethinyl oestradiol must be employed to ensure suppression. Progestogen is added on days 18 to 25 to produce a predictable withdrawal bleed. A serial formulation differs from a sequential one only in that placebo tablets, usually lactose, are used on the days when active tablets, are not taken. This minimizes failure from forgetting to take the tablet.

The other types of oral contraceptives shown in Fig. 16.12 are not yet available commercially, but they have certain useful properties and will be mentioned briefly.

The *incremental* type differs from the sequential preparation in that the oestrogen is started on day one permitting a lower dose to be used. An incremental increase of oestrogen is made with the addition of the progestogen on day 19 to 26. Two lactose tablets finish the course.

A tablet is therefore taken every day. A typical prescription for this type of pill consists of:

Tabs; ethinyl oestradiol, 0.05 mg daily for 19 days.

Tabs; ethinyl oestradiol 0.08 mg with norethisterone 0.5 mg daily for 7 days.

Tabs; lactose daily for 2 days.

Such a formulation may be manipulated to relieve first day dysmenorrhoea (see Chapter 15).

The *rotating* (alternating) pill uses continuous oestrogen for the full cycle, and a progestogen on days 21 to 28 to bring on a regular vaginal bleed. Cycle control is very satisfactory and progestogen effects are minimal. This preparation is often useful in management of women who experience headaches or migraine during oestrogen withdrawl of the other types of contraceptives (Fig. 16.12).

The *reversed sequential* type of contraceptive is desirable when oestrogen effects need to be reduced as much as possible, and vaginal bleeding is too irregular on a progestogen only "mini" pill. Breakthrough bleeding reduces after several cycles.

Other Hormonal Preparations

Progestogen injections, and implants act as the "mini" pill, or progesterone only pill.

In the *post-coital* pills high doses of oestrogens and progestogens within 72 hours of intercourse will interrupt implanation of the ovum. Hormonal side effects are very common, 25% of women experience nausea and vomiting.

Coital pills involve a single dose of progestogen taken 5 hours prior to coitus. This will give protection from fertilization until 18 hours after ingestion. If coitus occurs 12 or more hours after ingestion, sperm may survive causing fertilization.

Prostaglandins in effective doses can be used to produce an early abortion. They are thus abortifacients and not contraceptives. Side effects of diarrhoea and nausea make them unacceptable generally.

E. **Common side effects of oestrogen and progestogen**

Oestrogens:

1. *Nausea* – especially with high doses. It may also result in loss of libido and in depression.
2. *Leucorrhoea* – due to stimulation of cervical glands.
3. *Weight gain* – due to water retention.
4. *Impairment of lactation* – oestrogen-containing contraceptives ideally should not be used while breast-feeding.

5. *Leg cramps* – from water retention.
6. *"Menstrual migraine"* – due to withdrawal of oestrogens.
7. *Hypertension* – slight rise in pressure in majority of women, marked rise in a small percentage (Weir *et al*, 1974).
8. *Thrombosis* – risk exists but is very low. Morbidity is about 3 to 5 per million per year. Death due to motor car accidents are about 110 per million per year.
9. *Metabolic effects* – similar to those during pregnancy.

Progestogens:

1. *Weight gain* – nor-testosterone compounds stimulate appetite. In women with poor insulin reserves weight gain may result in diminished sugar tolerance.
2. *Breakthrough bleeding* – unless extra oestrogen is added.
3. *Recurrent vaginal infection* – with yeasts (monilia) due to decreased thickness of cornified lining of the vagina.
4. *Depression, chronic fatigue, irritability, loss of libido* – These develop slowly and may not be noticed during the first year on the medication. This is worse in women who previously experienced premenstrual depression.
5. *Reduction in menstrual flow* – due to arrest of endometrial growth.
6. *Acne* – antagonizes oestrogens and may accentuate acne in susceptible women.
7. *Chloasma* – This is facial pigmentation, especially with exposure to the sun. It develops slowly.
8. *Post-pill infertility* – 3/100 women 20 to 45 years of age ovulate irregularly for this reason. Following the use of oral contraceptives 1% of women are infertile due to ovulatory failure. Women with long cycles are more sensitive to hormones and run a higher risk (up to 4%) of ovulatory failure after withdrawal of the hormones.

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CHAPTER 17

ABNORMAL BLEEDING IN NON-PREGNANT FEMALES

General Instructional Objective

Understands the causes and treatment of abnormal bleeding in the non-pregnant female so that he can institute the appropriate management.

Specific Behaviours

1. Describes the physiology of the menstrual cycle.
2. Discusses abnormal hormonal production and the mechanisms by which it causes abnormal bleeding.
3. Discusses the causes of abnormal bleeding in the non-pregnant woman.
4. Discusses his provisional diagnosis of bleeding in the non-pregnant woman.
5. Discusses the management of a non-pregnant woman with vaginal bleeding.
6. Counsels non-pregnant women who present with problems of abnormal vaginal bleeding.
7. Explains the pharmacology of and indications for the common oral hormone preparations used for the treatment of abnormal vaginal bleeding in the non-pregnant woman.

Abnormal bleeding in non-pregnant females

1. *Cyclical changes* in the endometrium – the menstrual cycle.
2. *Cyclical changes* in the genital tract and breasts.
3. *Abnormal bleeding patterns* (definitions).
4. *Causes of abnormal uterine bleeding.*

5. *Diagnostic procedure.*
6. *Some diagnostic indicators.*
7. *Dysfunctional uterine haemorrhage.*
8. *Intermenstrual bleeding.*
9. *Postmenopausal bleeding.*
10. *Counselling a woman with abnormal bleeding.*

1. Cyclical changes in the endometrium – the menstrual cycle

An understanding of the hormonal control of the normal menstrual

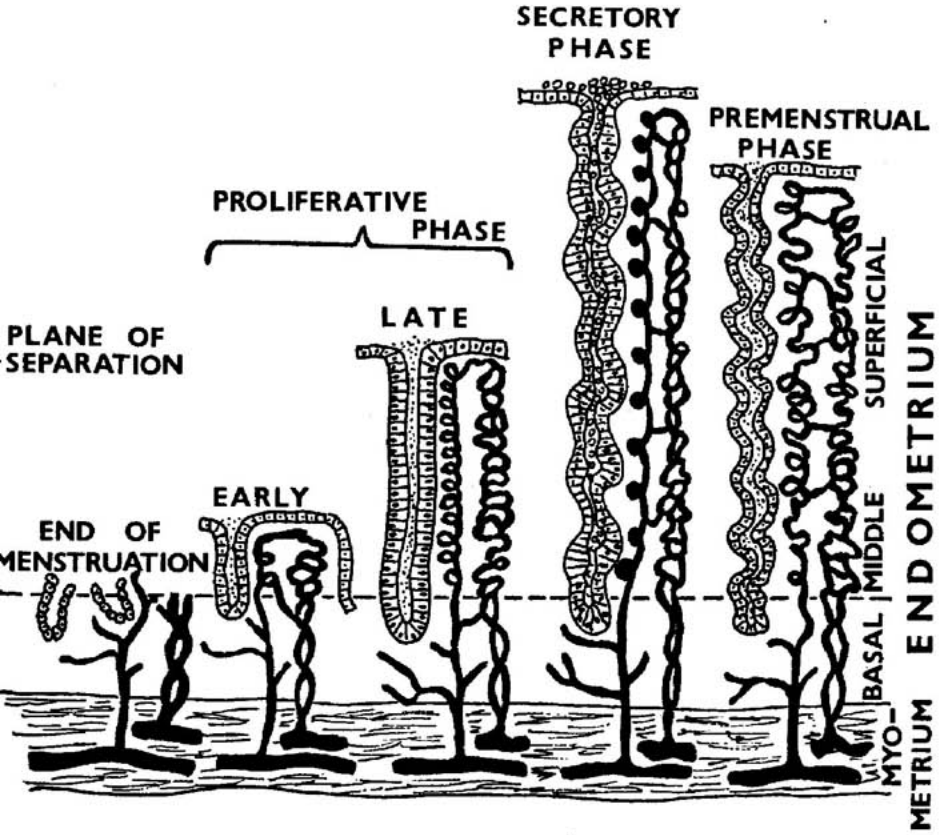

Fig. 17.1. The endometrial cycle.

cycle is essential to the rational management of disturbances of menstruation.

The changes that occur during the menstrual cycle are governed by the production of oestrogen and progesterone from the ovary. Ovarian function is controlled by pituitary gonadotrophins the release of which is regulated by the releasing hormones secreted by the hypothalamus. The activity of the hypothalamus is modified by other centres in the central nervous system, including neural areas which are influenced by the circulating level of oestrogen and progesterone.

The hormones involved, their cyclical production, and controlling factors are discussed in Chapter 16.

The Endometrial Cycle

The cycle involves –a. menstruation (menstrual phase),
b. a proliferative phase,
c. a secretory phase,

and classically lasts for 28 days (Fig. 17.1).

a. *The menstrual phase:*

With involution of the corpus luteum there is a fall in the concentration of oestrogen and progesterone and consequently shrinking of the endometrium.

The spiral arteries supplying the superficial and middle layers of the endometrium shorten, by tightening their coils. The loss of blood from the tissues and the compression of the spiral vessels, accompanied by intermittent constriction and relaxation of their bases, causes the endometrium to become increasingly hypoxic. Patchy ischaemic necrosis follows. Haemorrhage from already weakened spiral arteries further disrupts the tissues with separation and shedding of the superficial and middle layers of the endometrium (the menses) into the uterine cavity. The basal layer (and its straight arteries) are left intact.

The menses are discharged from the uterus by uterine contraction – the whole process taking 3-5 days.

b. *The proliferative phase:*

Even as areas of necrotic endometrium are being shed re-

generation of other areas is taking place from the disrupted bases of the glands in the basal layer. Within 3 days the surface is intact – with cuboidal cells lining straight glands and only about 1 mm. in thickness.

Under the influences of oestrogen the endometrium thickens to about 3 mm; the epithelium becomes columnar with basal nuclei and the stroma shows an increased water content, vascularity, protein content, and enzyme activity.

c. *The secretory phase:*

This begins 14 days before the onset of the next menstruation and coincides with ovulation. This time is constant and any variation in cycle length is due to variation in the preceding two phases.

During the secretory phase there are changes in the glandular and stromal elements of the endometrium.

Glandular changes include–

i. An increase in the number and dilatation of glands.
ii. Vacuolation of epithelium – initially occurs basally, displacing the nucleus to a more superficial site. Later (approximately 5-6 days of this phase) vacuoles appear on both sides of the nucleus.
iii. Secretion after approximately 7 days, rich in mucus and glycogen which will provide nutrition for a fertilised ovum.

In the stroma the cells accumulate fluid, glycogen, and lipids, and are separated by oedema. The spiral arteries enlarge and become increasingly coiled.

About five days prior to menstruation the changes are maximal, the endometrium reaching 5-7 mm in thickness. There is no further growth. Soon dehydration begins and the endometrium shrinks.

2. Cyclical changes in the genital tract and breasts

a. *Myometrium*

During the follicular phase of the menstrual cycle uterine contractions are stimulated by oestrogen which produces myometrial activity of high frequency and low amplitude.

Progesterone decreases the frequency and increases the amplitude of uterine contraction. This effect is probably mediated via prostoglandins.

b. *Cervix*

i. *Oestrogen*

1. Increases the quantity of cervical mucous.
2. Decreases the consistency of cervical mucous.
3. Facilitates sperm penetration of the cervical mucous plug at the time of ovulation.
4. The presence of NaCl and KCl results in the formation of fern-like crystals in dried cervical mucous.

ii. *Progesterone*

1. Antagonises the action of oestrogen and causes the production of a small amount of thick mucous.
2. There is no fern pattern when progesterone is dominant.

c. *Vagina*

i. *Oestrogen*

1. Stimulates cornification of superficial vaginal cells.
2. Increases glycogen deposition in these cells and consequently enhances lactobacillus growth, resulting in a low vaginal pH (about 4.4).

ii. *Progesterone*

1. Reduces the cornification index.
2. Predisposes to monilial infection (if influence is maintained for long periods).

Note: The cornification index – the percentage of cornified cells in a smear taken from the vaginal wall. It reflects the biological activity of circulating oestrogens.

d. *Breasts*

i. *Oestrogen* – Enhances growth of the duct system.
ii. *Progesterone* – Stimulates development of the alveoli.

3. Abnormal uterine bleeding patterns

a. *Menorrhagia* (hypermenorrhoea) is cyclical bleeding at normal intervals which is excessive in amount or duration. Its cause

is probably uterine (e.g. fibroid, adenomyosis) but may rarely be of a general nature such as a coagulation defect.

b. *Polymenorrhoea* (epimenorrhoea) is cyclical bleeding normal in amount but occurring at too frequent intervals. The cause is probably functional (ovarian).

c. *Polymenorrhagia* (epimenorrhagia) is cyclical bleeding which is excessive in amount or duration and occurs too frequently. This type of irregularity is often due to both a uterine and ovarian disturbance, e.g. chronic pelvic inflammatory disease.

d. *Metrostaxis* (metrorrhagia) is acyclical bleeding of any amount of duration. Usually ovarian dysfunction is responsible but there may be uterine or cervical lesions present such as carcinoma or polyps.

It is important to realise that abnormal uterine bleeding is not a disease entity *per se* but only a symptom for which a cause must be determined.

Note: Dysfunctional Uterine Bleeding. Abnormal bleeding occurring during the reproductive years unrelated to organic pathology. The pattern of bleeding is not specific.

4. **Causes of abnormal uterine bleeding**

a. *Pregnancy states–*

i. threatened or imcomplete miscarriage,
ii. ectopic pregnancy,
iii. hydatidiform mole,
iv. implantation haemorrhage.

b. *General medical conditions* – usually very rare.

i. coagulation defects,
ii. excessive capillary fragility,
iii. hypothyroidism,
iv. psychological upsets,
v. chronic liver disease,
vi. acute febrile illnesses.

c. *Pelvic pathology-*

i. *Neoplastic*

1. *Uterine* – malignant ulcerating carcinoma (corpus uteri, cervix), benign (polyps, submucous fibromyomata).
2. *Ovarian* – cysts or tumours producing oestrogen (may be benign or malignant, functional or apparently inert).

ii. *Inflammatory*

1. Salpingitis,
2. Pelvic cellulitis,
3. Endometritis.

iii. *Adenomyosis*

iv. *Increased endometrial thickness* – a chronic endometrial hyperplasia.

v. *Local injury*

1. intrauterine contraceptive devices,
2. insertion of tampons,
3. attempts to induce abortion,
4. neglected vaginal pessaries,
5. tears sustained during coitus.

iv. *Congenital abnormality* – uterus bicornis.

(Reference should be made to the chapters relevant to the above conditions for specific clinical features and management).

5. **Diagnostic Procedure**

a. *History*

i. amount, duration and frequency of haemorrhage,

ii. relationship to puberty, pregnancy and last normal menstrual period,

iii. full history involving relevant features of either general or local disorders,

iv. associated symptoms,

v. taking of any pills, particularly hormones.

b. *Physical examination*

i. general – evidence of anaemia,
ii. abdominal – for masses or fluid,
iii. vaginal – note tenderness; uterine or adnexal masses.

c. *Special Investigations*

i. cervical smear and vaginal cytology,
ii. blood tests – Haemoglobin,
White cell count,
ESR,
Bleeding time (where indicated).
iii. Examination under anaesthesia.
iv. *D and C* (premenstrually) and histology on scrapings.
v. Hysterosalpingopram – when abnormal bleeding persists after the above and no cause elucidated – a polyp or submucous fibroid may be missed when performing a curette.

6. Some diagnostic indicators

a. *If the bleeding occurs*

i. before 20 years of age – usually functional,
ii. 20-40 years – usually organic (pregnancy must be excluded),
iii. after 40 years, – exclude malignancy, often functional,
iv. postmenopausal – exclude malignancy-may be due to exogenous oestrogens.

b. *Polyps*

i. mostly asymptomatic,
ii. symptoms usually indicate ulceration and then include menorrhagia,
intermenstrual bleeding,
iii. definitive diagnosis–
by D & C or
hysterosalpingogram only.

c. *Fibromyomata*

i. may be asymptomatic,
ii. gradual, progressive menorrhagia,
iii. pressure symptoms–
constipation,
frequency,
lower extremity oedema,
iv. may cause intermenstrual bleeding if ulcerated,
v. may be palpable bimanually,
vi. history of infertility, abortion.

d. *Chronic pelvic inflammatory disease*

i. polymenorrhoea, polymenorrhagia,

ii. chronic pelvic ache ± sacral backache,
iii. dysmenorrhoea – especially premenstrually,
iv. deep dyspareunia,
v. vaginal examination discloses tenderness and induration of adexae with occasional masses.

e. *Endometriosis*

i. menorrhagia,
ii. dysmenorrhoea – especially if maximal on 2nd/3rd day of menstruation,
iii. progressive dysmenorrhoea over age of 30,
iv. deep dyspareunia,
v. infertility,
vi. bizarre sysmptoms.

f. *Carcinoma*

i. intermenstrual or post-coital bleeding,
ii. serous discharge,
iii. other symptoms late – indicate invasion of bladder, bowel or nerves.

7. Dysfunctional uterine haemorrhage (D.U.H.)

Once an organic basis for abnormal uterine bleeding is excluded, the diagnosis of dysfunctional uterine bleeding can be made. There is often a dysfunctional element associated with a pathological condition.

There are three types of dysfunctional uterine bleeding –

a. Anovular
b. Ovular
c. Pseudo-ovular

a. *Anovular*

D.U.H. is acyclical and occurs predominantly during the first 2-3 years after puberty and the 5 years prior to menopause. The onset of symptoms is often *abrupt*, and one manifestation is metropathia haemorrhagica or Schroeder's disease.

Under the influence of F.S.H. the Graafian follicle enlarges. However, there is no midcycle peak of LH (no releasing factor secreted) so the follicle neither ruptures nor becomes luteinised.

Progesterone is not produced and there is no secretory change in the endometrium.

The endometrium under the influence of unopposed oestrogen, hypertrophies and may become polypoidal. The prolonged high concentrations of oestrogen may produce a short period of amenorrhoea. Eventually the thickened endometrium cannot be maintained by relatively inadequate levels of oestrogen and is *shed irregularly*. The subsequent *bleeding* is *prolonged or heavy but painless* – an example of oestrogen withdrawal bleeding.

Histologically the endometrium is thick and with a great increase in epithelial cells the uterine glands undergo cystic dilatation.

Sometimes a continuous but relatively low level of oestrogen production results in oestrogen breakthrough bleeding. The endometrium is thin and histologically resembles a normal proliferative phase.

b. *Ovulatory* cycles may be associated with abnormal bleeding patterns. Commonly *polymenorrhoea* occurs due to shortening of the proliferative phase of the cycle. With persistence of the corpus luteum *slight bleeding follows* a normal menstruation as the endometrium is shed irregularly.

Low grade pelvic infections, often dating from pregnancy, may present in this way.

c. *Pseudo-ovulatory cycles* occur when there is luteinisation of an unruptured follicle due to secretion of a moderate but insufficient amount of LH to cause ovulation.

The luteal phase is short and histologically the endometrium is secretory.

Note: In all types of dysfunctional uterine bleeding there is no pathology detected on general or vaginal examination.

Treatment may include;

a. General measures
b. Hormonal therapy
c. Surgical treatment
d. Radiotherapy
e. Psychotherapy

a. *General Measures:*

If there is evidence of anaemia oral iron is necessary. It is also advisable in women having heavy periods, but not yet manifesting anaemia.

b. *Hormonal Therapy:*

i. The type of hormonal therapy instituted may depend on–

1. the age of the patient, and
2. the need for oral contraception.

In young patients, not exposed to the risk of pregnancy norethistrone (NE) 0.5 mg and ethinyl oestradiol (EE) 0.05 mg can be used during the third week of each cycle to produce regular shedding of the endometrium.

In younger women or women who have not yet completed their family, it is desirable to avoid using high doses of progestogen due to the risk of causing 'post-pill' infertility. Cycle control may be achieved with –

EE 0.05 mg (days — 1-19),

EE 0.05 mg + NE 0.5 mg (days 20-26).

Norethisterone may be used over a greater part of the cycle if at first not successful.

Alternatives include–

1. *Reversed sequential* oral contraceptive,

   e.g. lactose days 1-5,
   NE 0.5 mg days 6-16,
   NE 0.5 mg — EE 0.05 mg days 17-26,
   lactose days 27-28,
   and repeat.

   Note: i. Ethinyl Oestradiol may be used earlier in the cycle or in larger doses to control breakthrough bleeding.

   ii. Norethisterone may be increased if there is insufficient reduction in menstrual flow.

2. *Combined oral contraceptive*
   e.g. Minovlar ED –

NE 2.0 mg + EE 0.05 mg days 1-26
lactose days 27-28.

ii. *Treatment* of a bleeding episode – when organic pathology has been excluded.

EE 0.05 mg qid. till nausea develops.

It will take as long as 72 hours for this therapy to take effect. As most cases of heavy bleeding will resolve naturally within this time it is indicated only when bleeding is likely to continue for longer than 3 days.

The dose is reduced slowly to prevent oestrogen withdrawal bleeding. Following control of bleeding norethistrone must be added to ethinyl oestradiol to prevent excessive growth of the endometrium and the occurrence of a further episode of heavy bleeding.

iii. *Rationale of Hormone Therapy*

1. When menorrhagia is due to failure of ovulation the heavy blood loss can usually be controlled by supplying a progestogen to bring about a secretory change in the endometrium.
2. When oestrogen production is excessive the growth promoting effect of the oestrogen on the endometrium can be inhibited by giving norethisterone. One milligram of norethisterone can antagonise the proliferative effect on the endometrium of a moderately high dose of oestrogen.
3. In ovulatory cycles with menstrual symptoms the ovarian function is suppressed and menstrual flow is regulated by exogenous hormones. This can be accomplished by using ethinyl oestradiol from the first day of menstruation to suppress the secretion of FSH-RH. If this approach does not control the blood loss the duration of progestogen administration must be increased.

   If necessary the daily dose of norethisterone is also raised. The use of progestogen for large portions of the cycle increases the frequency of progestogen side effects. Breakthrough bleeding is more common unless there is an increase in the dose of oestrogen to prevent this.

c. *Surgical Treatment* – is instituted only if symptoms persist

despite medical treatment and are severe enough to require further treatment. As hysterectomy is involved the age of the patient, and desire for further children must also be taken into account.

Note: The curettage often results in cessation of symptoms – the reason for this is not understood.

d. *Radiotherapy* – is *rarely* indicated. 600r or 50 mg radium in the uterine cavity for 40 hours will produce a radiation menopause.

e. *Psychotherapy* – Emotional problems may underlie menstrual disorders especially in women of 20-40 years of age. The mechanism operates via the hypothalamus.

The patient should be encouraged to discuss her problems and their possible solutions. She must be left to make her own decisions – the doctor only provides technical or factual advice.

Hormonal therapy may be used as an adjunct to psychotherapy, to control bleeding temporarily.

8. **Intermenstrual Bleeding**

This is irregular vaginal bleeding associated with a normal menstrual cycle.

Causes:

a. *General* – as in Section 4 above.

b. *Local* –

- Carcinoma of cervix,
- Ulcerated uterine polyp,
- Ulcerated submucous fibromyoma,
- Intrauterine contraceptive device.

c. *Hormonal* – oestrogen withdrawal at midcycle, sometimes associated with pain (*mittelschmerz*).

Management:

The possibility of carcinoma of the cervix *must* be excluded.
The diagnostic procedure follows that already outlined.
Treatment is for the specific condition (see relevant chapters).

9. **Postmenopausal bleeding** – is bleeding following cessation of

menstruation. The menopause or, 'cessation of menstruation' is often considered as at least 6 months amenorrhoea in a woman over 40 years of age. Pregnancy must be excluded. Furthermore, post-pill suppression may occur for period of six months in as many as 1% of women who have used a high dose of progestogen preparation. The tendency is therefore to require at least 12 months amenorrhoea before assuming a woman is menopausal even then the only way to establish that she is menopausal is to demonstrate a persistently high blood level of LH, and/or failure of the ovaries to respond to stimulation by human pituitary gonadotrophin.

Causes:

a. *General* – as above.
b. *Local* –

i. Neoplastic

. uterine carcinoma,
. carcinoma of cervix (and other parts of genital tract),
. ulcerated polyps,
. ulcerated submucous fibromyomata.

ii. *Atrophic*

. senile vaginitis
or
. endometritis.

c. *Hormonal* –

. exogenous oestrogen administration,
. oestrogen secreting tumours of ovary,
. recurrence of ovular bleeding.

d. *Trauma* –

. proccidentia with ulceration,
. post irradiation.

e. *Bleeding from other sources* –

. urethra,
. bladder,
. rectum.

f. *Idiopathic.*

Management:

When bleeding occurs after the menopause the possibility of malignancy *must* be excluded, especially if the bleeding is continuous or recurrent. The most common cause of post-menopausal bleeding is the administration of oestrogens and evidence of this must be specificially sought. The diagnostic procedures are similar to those set out under Section 5.

Treatment:

Refer to chapters dealing with the specific disorders.

10. **Counselling a woman with abnormal uterine bleeding** –

requires careful investigation and then reassurance. Counselling should be aimed at patients who have a fear of malignancy. Always encourage the patient to discuss her symptoms, her fears, and her problems, and persuade her to attend for a regular check-up. Be prepared to explain fully and rationally what has caused the bleeding and how you intend to manage it.

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CHAPTER 18

BENIGN TUMOURS OF THE GENITAL TRACT

General Instructional Objective

Recognises and evaluates tumours of the genital tract so that appropriate management can be insituted.

Specific Behaviours

1. Demonstrates a knowledge of the common infective and benign tumours which may be found in the genital tract.
2. Differentiates between common infective and non-infective tumours of the genital tract.
3. Discusses the clinical features of benign tumours of the genital tract.
4. Makes and discusses a provisional diagnosis of a benign tumour of the gential tract.
5. Indicates the management of choice of benign tumours of the genital tract.

Benign Tumours of the Female Genital Tract

Benign Tumours of the vulva and perineum

A. **Epidermal Origin**

1. **Viral Warts** – Condyloma accuminata

Incidence:

Common. These occur mainly in women of childbearing age and are especially common during pregnancy.

Aetiology:

Viral infection, almost certainly veneral. They are often associated with vulvo-vaginal infection with discharge when trichomonas, candida albicans or gonococcus may be isolated.

Pathology:

Nearly always multiple, they occur as discrete papillary processes. These tend to spread and coalesce to form large papillary growths. Fresh lesions are pinkish-white, whereas older ones become brown like the skin. When viral warts occur as large flat moist lesions on the perineum and peri-anal skin they are termed condyloma accuminata.

Histologically they consist of thickened epithelium thrown into folds around connective tissue stalks.

Malignant change has been reported but is very rare.

Clinical Features:

Location is mainly on the skin of the labia majora, perineum and peri-anal skin. They may involve the vagina. Alone, the warts may cause a foul-smelling discharge. Usually, however, they are either asymptomatic or the symptoms of the associated vaginitis predominate.

Treatment:

i. Application of 20% Podophyllin in tincture of Benzoin to the wart alone (avoid skin). The warts blanch then slough off in 3-4 days. Repeated application may be needed for larger lesions or fresh warts.
ii. If the warts are large and extensive, diathermy of the major ones may be necessary.
iii. In pregnancy treat the associated vaginitis if present, and leave definitive treatment of the warts until after delivery. (They may disappear spontaneously.)

2. **Cutaneous Cysts** (Sebaceous and Epidermal)

Incidence: Common.

Etiology: Developmental.

Pathology:

Epidermal cysts – contain keratin within the cyst which is lined by stratified squamous epithelium.

Sebaceous cyst – contain sebum and keratin (often foul smelling) and are lined by stratified squamous epithelium with many foamy plump sebaceous-type cells.

Clinical Features:

Both types occur as firm, dome-shaped cysts with a central punctum. Usually they present as a cosmetic problem but occasionally the cysts may discharge or become infected causing symptoms.

3. *Others*

. Seborrheic keratosis.
. Squamous papillomata.
. Pigmented naevi (be alert to signs of malignant change).
. Sweat gland tumours.

B. Mesodermal Tumours

These are rare as a cause of symptoms. Following is a list of the common ones:

. Fibromas.
. Lipomas.
. Neurofibromas.
. Leiomyomas.
. Haemangiomas.
. Lymphangiomas.

Other Vulval Swellings

1. **Bartholin's Cyst and Abscess**

Incidence:

Fairly common.

Etiology:

A Bartholin's cyst arises due to obstruction of the duct of a Bartholin's gland following infection or trauma. The duct then becomes distended with glandular secretions.

This cyst may become infected, the common organisms involved being E. Coli, then Streptococci, Staphylococci and Gonococci.

Pathology:

The cyst wall is lined by the transitional epithelium of the duct. Glandular secretions filling the cyst are replaced by pus if infection occurs.

Clinical Features:

Cyst: This occurs as a cystic swelling deep to the posterior part of the labium just external to the hymen at the 5 or 7 o'clock positions. It usually does not exceed 5 or 6 cm. in diameter. Few symptoms arise from the cyst but the patient may either present because she believes it is something serious, or for costmetic reasons.

Abscess: When the cyst becomes infected the swelling becomes extremely painful and tender. The patient may even find it difficult to walk or sit. The infection may subside, become recurrent, or become chronic in which case a firm, slightly tender mass will be present.

Treatment:

Incision or spontaneous rupture of the abscess cause rapid relief of symptoms but unless definite treatment is performed frequent recurrence may occur. The definitive treatment is excision of the edges and marsupialisation. An incision about 2-5 cm. long is made over the vaginal aspect of the cyst or abscess along the line of the labium minus. The cavity is drained and the lining sutured to the skin. This treatment preserves the function of the gland and recurrences are unlikely.

2. **Urethral Caruncle**

Incidence:

Fairly common in post-menopausal women.

Etiology:

Atrophic vaginitis with shrinking of the vaginal epithelium causes traction on the posterior urethral membrane which undergoes eversion or prolapse.

Pathology:

It may be polypoid, angiomatous, or granulomatous.

Histologically, the lesion is covered by stratified squamous or transitional epithelium with deep crypts due to infolding. On section these crypts appear as epithelial islands deep in the stroma and may simulate malignant invasion.

Clinical Features:

The caruncle usually presents as a cherry red blob at the posterior urethral margin. It is often asymptomatic. If symptoms occur they may include pain or discomfort (especially superficial dyspareunia) and occasional blood staining. If infection occurs the caruncle becomes exquisitely tender.

Treatment:

Only treat if symptoms occur. The definitive treatment is diathermy, excision of the caruncle and the area of urethra from which it arises. The lesion tends to recur.

NB: Differentiate urethral caruncles from urethral prolapse (of whole urethral circumference) which can occur in any age group including children, following straining or acute urinary tract infection.

3. **Genital Prolapse**

The diagnosis of this condition will be obvious on examination.

4. **Stenson's Duct Cysts**

These occur in relation to the urethra and have a simular etiology to Bartholin's Cysts.

Benign Tumours of the Vagina

1. **Gartner's Cyst**

Incidence:

Seen occasionally in the out-patient's department.

Etiology:

It is derived from remnants of the embryonic Wolffian Duct. This duct runs alongside the vagina at an early stage in development but normally regresses totally.

Pathology:

Gartner's cysts are most commonly found as single lesions in the upper portion of the vagina. They are usually thin walled, small and insignificant.

Clinical Features:

The lesion rarely gives rise to symptoms. It is best left alone. Attempted removal may lead to a deep, bloody and dangerous dissection during which the bladder or ureter or both may be injured.

2. **Vaginal Adenosis**

Incidence: Rare.

Etiology: Developmental.

Pathology: The normal squamous epithelium covering the vagina is replaced by columnar and gland bearing epithelium. Malignant change has been reported.

Clinical Features:

The vagina and possibly also the cervix are covered by cysts up to several mm. in diameter. There is excessive mucous secretion.

Treatment:

This is difficult. The excision is extensive and skin grafting is needed.

3. **Others**

Any benign epithelial or connective tissue tumour may occur but it is very uncommon and only rarely produces symptoms.

Benign Tumours of the Cervix

1. **Endocervical Polyps**

Incidence: Common.

Etiology: Not known.

Pathology:

These occur as bright red, friable, vascular growths which are usually pedunculated and arise in the endocervical canal.

They may be single or multiple. Usually they are no more than a few mm. in diameter but rarely may reach 3 cm.

Histologically they are of two basic types:

i. Mucous polyps – Consist of cervical mucous glands, somewhat distended, in a vascular bed of loose fibrous tissue and covered usually by columnar but sometimes squamous epithelium. Inflammation and ulceration of the apex is common.
ii. Fibro-epithelial polyps. These lack the mucous glands.

Clinical Features:

i. The most common symptom occurs with mucous polyps and consists of a mucous or mucopurulent vaginal discharge.
ii. Contact bleeding may be produced (e.g. post coital).
iii. Intermenstrual bleeding may occur, especially after coughing or straining.

Management:

i. If abnormal bleeding has occurred, a diagnostic D & C is mandatory, to exclude malignancy. Prior to the curettage any observable polyps may be removed by twisting their pedicle with sponge holding forceps. During the D & C the cervical canal may be curetted to remove any additional polyps which may be there.
ii. Even in the absence of symptoms it is best to remove visible polyps. This may be performed as a simple out-patient procedure.
Cautery may be needed for haemostasis or if the discharge is the main problem and there is associated ectopic columnar epithelium.

2. **Nabothian Follicles**

These are minute (1-5 mm. diameter) follicles which may be present on the vaginal surface of the cervix as small nodules. They are probably the remnants of cervical glands which remain when ectopic columnar epithelium undergoes squamous metaplasia. They contain mucus (not pus), do not produce symptoms and require no treatment.

3. **Ectopic Columnar Epithelium** (also termed "Cervical Erosion")

This is *not* a tumour but is conveniently discussed here because of its frequent confusion with malignancy.

Incidence:

It occurs on about 10% of cervices. The incidence (and extent of individual lesions) increases during pregnancy, during use of oral contraceptives and with increasing parity. It is rare after the menopause.

Etiology:

Ectopic columnar epithelium (E.C.E.) is an outgrowth of endocervical epithelium which may occur under oestrogenic stimulation. Metaplastic and infective causes are unlikely.

Pathology:

Ectopic columnar epithelium is bright red due to the presence of only a single layer of epithelial cells over the vascular stroma. It appears "velvety" and being very friable bleeds easily.

Clinical Features:

i. Increased vaginal discharge may be present.
ii. Contact bleeding (e.g. post-coital).

Treatment:

Only if symptomatic.

i. If bleeding has occurred, exclude malignancy. This may be done by Pap. smear, colposcopy and biopsy, and diagnostic D. & C.
ii. If intractable discharge is present first withdraw any exogenous oestrogen stimulation, and if this fails, proceed with cautery of the lesion.

Differential Diagnosis:

. Ectropion – produced by laceration of the cervix.
. Cervical carcinoma.

4. Chronic Cervicitis

This diagnosis is usually incorrectly applied to E.C.E. which is producing leucorrhoea. It should not be diagnosed unless inflammatory changes in the cervix are associated with an increase in muco-purulent discharge tenderness or inflammatory fixation to surrounding tissue.

For more details see the chapter on "Leucorrhoea and Pelvic Infection".

Bengin tumours of the uterus

1. Uterine Polyps

Incidence: Common.

Etiology:

i. Adenomatous (endometrial) polyps may be part of a general endometrial hyperplasia – e.g. associated with exogenous oestrogen in a post-menopausal woman.
ii. The cause of adenomatous polyps unassociated with endometrial hyperplasia, and the cause of adenofibromatous polyps is unknown.
iii. Placental polyps are small organised pieces of retained placental tissue.

Pathology:

Uterine polyps are usually small red or pinkish-white, pedunculated tumours projecting from the endometrial surface.

Histologically:–

a. Adenomatous polyps – These consist of endometrial stroma and glands covered by a single layer of columnar epithelium.
b. Adenofibromatous polyps – These are differentiated by the presence of fibrous stroma. They are more common post-menopausally.
c. Placental polyps – These may be difficult to distinguish from chorioncarcinoma.

Clinical Feautres:

i. As a part of endometrial hyperplasia, they may be associated with menorrhagia or metropathia haemorrhagica.
ii. They may cause intermenstrual or post-menopausal bleeding. If a polyp extrudes from the cervix it may cause post-coital bleeding or may be discovered in the vagina by the patient.

Management:

If the presenting symptom is abnormal vaginal bleeding it is mandatory to do a diagnostic D & C to exclude malignancy. The polyps may be discovered during this procedure and their removal is facilitated by the use of small sponge forceps. All tissue removed should be examined by a pathologist to exclude malignant change.

2. **Uterine Fibroids** (Fibromyomas)

Incidence:

This is the most common tumour in the female. 20% of women over 30 years have at least one fibroid in the uterus.

Etiology:

This is unknown. Oestrogens have been implicated. The following list of facts may be helpful:–

i. Many enlarge rapidly during pregnancy or with exogenous oestrogens.
ii. They are more common in women who have not borne children. (A result, or cause of relative infertility)
iii. There are racial factors in their incidence. They are more common in Negroes in whom they tend to occur at a younger age.
iv. Tissue culture experiments indicate smooth muscle cell origin.
v. They atrophy, with the uterus, after menopause.

Pathology:

Fibroids most commonly arise from the body of the uterus. They may be single or multiple. Usually they start off as firm round intramural tumours, but with growth, and under the effects of uterine contractions they may become extruded from the wall to become either submucous or subserous fibroids. Submucous fibroids which are initially sessile may become polypoidal in the uterine cavity and if the pedicle is long enough they may tort. Subserous fibroids may similarly become pedunculated. In size, they may vary from microscopic tumours to ones that may fill the abdomen. Fibroids are enclosed in a false capsule of compressed uterine muscle, an important point in differentiating them from other tumours such as adenomyomas. On transverse section they are pale with a characteristic whorled appearance.

Histologically fibroids are composed of smooth muscle cells arranged in bundles in a whorled pattern with a variable amount of fibrous tissue. They have a blood supply which enters from the periphery but are relatively avascular. As a result degenerative changes may occur. These may take the form of:–

i. Hyaline degeneration – This occurs to some degree in most fibroids. Larger areas may liquify leading to cystic degeneration.
ii. Cystic degeneration.
iii. Calcification – Occurs especially in pedunculated tumours (poorer blood supply).
iv. Necrobiosis or red degeneration – Usually occurs during pregnancy or after the menopause but may occur at any time. It is due to alterations in vascularity with consequent infarction which may be diffuse or focal. Histologically there is loss of the fibromyomatous pattern and loss of nuclei.
v. Necrosis – Occurs particularly after other degenerative changes.
vi. Infection – This is rare. It may occur for example during or after appendicitis with the formation of adhesions between the fibroid and the bowel. It can also be associated with salpingitis.
vii. Sarcomatous degeneration. This is very rare. If it occurs the fibroid typically has a soft homogenous area. It is highly malignant and locally invasive with early blood-borne metastases to the lungs.

Clinical Features:

Most are asymptomatic. Symptoms are rare before the age of 25, the greatest incidence of symptoms being between 30 years and the menopause after which they usually atrophy.

i. Abdominal swelling – This is a common form of presentation. It occurs only if the tumour is very large. If pedunculated the tumour may be felt to move.
ii. Pressure effects – Pressure on the pelvic veins may cause

oedema and/or varicose veins in one or both legs, or it may cause haemorrhoids. Pressure on the bladder may result in frequency of micturition or stress incontinence. If the fibroid is present in a retroverted uterus it may become incarcerated in the pelvis and produce urinary retention. Fibroids do not cause constipation. An extremely large fibroid may cause dyspnoea due to the increased abdominal pressure acting on the diaphram.

iii. Progressive menorrhagia – This may occur in submucous fibroids because:–
   a. Increase in surface area of endometrium.
   b. Increased vascularity.
   c. Formation of endometrial "polyps" at the angle between the submucous fibroid and the uterine wall (areas of endometrium are not completely shed with each menstrual flow). Intermenstrual bleeding occurs only with ulcerated fibroids or fibroid polyps.

iv. Infertility – This might be due to uterine distortion or mechanical obstruction of the Fallopian tubes or there may be no obvious reason.

v. Cause of abortion-Fibroids are often implicated and it is reasonable that implantation in relation to a submucous fibroid could lead to abortion. However, many prenancies go to term with uteri grossly distorted by fibroids.

iv. Changes in pregnancy – Fibroids may increase in size or may become less evident due to softening and oedema. Certain degenerations especially red degeneration are more common during pregnancy. A cervical fibroid (very rare) or a fibroid in the lower part of the uterus may cause obstructed labour. If a fibroid interferes with proper uterine retraction it may cause a post-partum haemorrhage.

vii. Pain –
   a. Congestive dysmenorrhoea due to increased vascularity.
   b. Constant backache may be caused by moderately large fibroids in a retroverted uterus.
   c. Acute or subacute pain may occur with torsion of a pedunculated fibroid or due to red degeneration.
   d. Uterine colic may be associated with attempts by the uterus to expel a fibroid polyp.
   e. Infection and sarcomatous degeneration are rare but may cause pain.

viii. Vaginal discharge – This is rare as a symptom of fibroids and usually reflects ulceration or degeneration of a submucous fibroid.

ix. Secondary symptoms such as anaemia and urinary tract infection may occur.

Signs:

The most distinctive feature of fibroids is their consistency. They are palpated as rounded, smooth and firm swellings.

Management:

Management of the individual case depends on severity of symptoms, age, parity, general health of the patient and the site and size of the tumour.

i. Conservative treatment – This is employed when the fibroid is small, the diagnosis certain and there are no symptoms. It is also used during pregnancy. Such patients should be re-assessed regularly. Larger fibroids may be managed in this way near the menopause in anticipation of atrophy as long as they are not producing symptoms and the diagnosis is certain.

ii. Surgical Treatment – Indications for surgery are-:
   a. Doubtful diagnosis – suspicion of malignancy.
   b. Fibroids larger than a "12 week" sized uterus.
   c. Fibroids causing symptoms.
   d. Infertility attributable to no other cause.
   e. Other pelvic or abdominal disease warranting surgery anyway.
   f. Presence of a fibroid in a non-pregnant woman, which may obstruct labour, if she becomes pregnant.

If there is potential and desire for childbearing a myomectomy is preferred. This operation may be associated with the complications of adhesion formation and intestinal obstruction. However, uterine rupture during a subsequent pregnancy is very rare.

Alternatively, hysterectomy may be performed. This is associated with less haemorrhage and removes the possibility of fibroids recurring. Hysterectomy may be performed abdominally or vaginally.

The Differential Diagnosis of Fibroids (or of any pelvic mass with or without symptoms – especially abnormal bleeding):

1. **Pregnancy**

This must always be excluded whenever a pelvic mass is present with or without symptoms.

Distinguishing Features:

i. Symetrical uterine enlargement. Fibroids usually produce asymetrical enlargement.
ii. Positive pregnancy test.
iii. Echography – will reveal the cause of enlargement.

Rarely a single large fibroid may simulate pregnancy. If it causes abnormal bleeding it may then be confused with a miscarriage. It is possible to mistake the pregnant uterus for a fibroma, a mistake which should never occur.

2. **Carcinoma**

This is the second condition which must be excluded whenever an abnormal pelvic mass, pelvic pain, or abnormal vaginal bleeding occurs.

Distinguishing Features:

i. Rapidly progressive onset.
ii. Abnormal vaginal bleeding. This makes a diagnostic D & C mandatory.
iii. Systemic symptoms – Loss of weight, malaise, etc. Some malignancies (e.g. Fibrosarcoma of uterine wall, or an ovarian carcinoma) will not be discovered by D & C and if any doubt as to the diagnosis exists then a laparotomy is indicated.

3. **Endometriosis and Adenomyosis**

Distinguishing Features:

i. Endometriosis mainly produces severe dysmenorrhoea which is not a feature of fibroids.
ii. If adenomyosis causes uterine enlargement it is symetrical and firm, not asymetrical and hard as with most fibroids.

4. **Ovarian Tumours**

Distinguishing Features:

i. Commonly cystic.
ii. Usually not continuous with the uterus.
iii. Calcification patterns are usually different (onion ringing in fibroids, more haphazard in ovarian fibromas).

Confusion between the two tumours is most likely to occur in a patient who is known to have fibroids. In such a case fluctuation may be misinterpreted as cystic change. Also, if adhesions connect the ovarian mass to the uterus, then differentiation will be made even more difficult. Alternatively, a large pedunculated fibroma may appear to be in discontinuity with the uterus. Differentiation in some cases may not be possible until laparotomy is performed.

5. **Hydrosalpinx and Pyosalpinx**

Distinguishing Features:

i. Recent or current history of foul vaginal discharge, or exposure to venereal disease. Alternatively there may be a history of abortion or recent delivery.
ii. Recent or current history of steady or spiking fever.
iii. Local peritoneal inflammation.
iv. Presence of a yelllow, purulent discharge from the cervix and palpation of the fornices.
v. Severe pain elicited by movement of the cervix and palpation of the fornices.
vi. Elevated white cell count (20-25,000/cu. mm.)
vii. Swabs from cervix may show intra-cellular Gram negative diplococci on microscopic examination.

NB: Hysterosalpingogram is contraindicated if active pelvic inflammatory disease is suspected.

6. **Pelvic Abscess**

Distinguishing Features:

i. May be recent history of pelvic or abdominal surgery.
ii. Presence of spiking fever and moderate leucocytosis.
iii. Fluctuation of the mass on palpation.
iv. Tenderness on palpation.
v. Commonly located in dependent situation, i.e. Pouch of Douglas.

If there is no history to suggest a pelvic abscess and the woman is over 30 with known fibroids the occurrence of a pelvic abscess may be mis-diagnosed as degeneration in a fibroid. However, overall assessment of the factors outlined above should alert one to the diagnosis.

7. **Pelvic Tuberculosis**

   This is rare in Australians but in migrants it is quite common. About 5 % of migrant women presenting with infertility have pelvic tuberculosis.

   It reaches the female genital tract via the blood stream from a pulmonary lesion involving initially the tubal submucosa. From here it may spread along the lumen of the Fallopian tube to involve the endometrium. The endometrial lesions are shed each month during menstruation.

   If pelvic tuberculosis presents as an adnexal mass it may be confused with a pelvic tumour. It may also present because of infertility or abnormal P.V. bleeding.

   Distinguishing Features:

   i. Mantoux test – May be positive. A negative result may exclude tuberculosis.
   ii. Chest X-ray.
   iii. D & C – with guinea pig inocculation and microscopic examination.

   Hysterosalpingogram is contraindicated if active pelvic tuberculosis is suspected.

Benign Tumours and Cysts of the Ovary

Because of their clinical implications and the frequency of maliganant change these are discussed on the chapter on "Gynaecological Malignancy".

Summary - Assessment and Diagnosis of Pelvic Tumours

1. History and examination.
2. Pap. smear – routine.
3. Diagnostic D & C. This is mandatory if abnormal bleeding has occurred from the vagina after a pregnancy state has been exlcuded.
4. Radiological investiagtion–
   a. Plain X-ray.
   b. Hysterosalpingogram – Contraindicated if active pelvic inflammatory disease is suspected.
5. Haemoglobin – White Cell count, E.S.R.

6. Laparotomy – If diagnosis from above investigation is equivocal and malignancy is a possibility.

CHAPTER 19

GYNAECOLOGICAL MALIGNANCY

General Instructional Objective

Recognises the presence of and understands gynaecological malignancy so that he can initiate and participate in continuing management.

Specific Behaviuors

1. Discusses the clinical differences between benign and malignant tumours of the genital tract.
2. Discusses the clinical features and course of the various types of gynaecological malignancies.
3. Indicates a knowledge of the possible aetiological factors involved.
4. Indicates a knowledge of the various diagnostic techniques.
5. Indicates the available methods of treatment and discusses the prognosis.
6. Discusses problems of counselling women with gynaecological malignant disease.

Gynaecological Malignancy

In this chapter the malignant tumours of the genital tract will be discussed. Benign ovarian tumours will also be included. Breast cancer, the most common gynaecological malignancy will not be dealt with here.

Genital cancer is second only in frequency of incidence to breast cancer. It includes several anatomical regions the relative importance of which are shown in Table 19.1. By far the most common malignancy is carcinoma of the cervix uteri, and its incidence in the U.S.A. has been noted to be 44 per 100,000 women. It is obvious then, that although an important entity it is nevertheless relatively common in clinical practice.

	Percentage of all carcinoma	Percentage of genital carcinoma
Cervix uteri	11	60.25
Corpus uteri	6	25.0
Ovary	5	10.0
Vulva	1	3.0
Vaginal	–	1.0
Chorion	–	0.5
Oviduct	–	0.25

Table 19.1 The distribution of genital cancer. (From Llewellyn-Jones 1973).

A. Clinical Features of Genital Malignancy

The presence of the following features during a clinical examination should arouse one to suspect a malignancy and to investigate the patient further.

1. *Cervical Carcinoma* – Age over 40 years.
   - Important symptoms include post-coital bleeding, unexplained vaginal spotting or bleeding or a foul vaginal discharge.
   - Pelvic pain, leakage of urine and faeces from the vagina are often signs of advanced disease as is weight loss and anorexia.
   - On palpation of the cervix, hardness, friability, fixation, and bleeding are characteristic of malignancy.
2. *Uterine Malignancy* – Age over 50 years.
   - Irregular bleeding (especially post-menopausal), discharge, and pain. The pain is hypogastric and is not severe, but occurs for 1-2 hours at about the same time each day.
3. *Ovarian Malignancy* – Age over 40 years.

   - Symptoms are generally absent until a late stage of the disease. The vague abdominal discomfort with dyspepsia that may occur early, is too non-specific to be followed up.
   - Physical features may include an ovarian enlargement of more than 6 cm. diameter (25% chance of malignancy post menopausally), rapid growth of an ovary or of an abdominal swelling; ascites; solid bilateral ovarian swelling; and a multinodular, solid ovary.
   - Late symptoms may be those of a dull, aching, pelvic pain, frequency of micturition, and ankle swelling. Irregular or post-menopausal bleeding is occasionally noted.
4. *Vulval Malignancy* – Age over 50 years.
   - The presenting complaint may be pruritis, local pain, pain on micturition, or a blood-stained discharge.
   - There may be a lump or a painful ulcer that is hard, friable, and bleeds easily.
   - Lesions such as leukoplakia, kraurosis, diabetic vulvitis, and venereal disease may precede the malignancy.
5. *Vaginal Malignancy* – Age over 50 years.
   - Vaginal discharge and spotting, pain, urinary symptoms, and groin masses are the major symptoms.
   - The presence of a palpable or visible lesion is the main physical finding. It may be an ulcer with a hard base and raised edges, which bleeds on touching.
6. *Tubal Malignancy* – Usually post-menopausally.
   - Pelvic pain accompanied by irregular vaginal bleeding and discharge, frequently described as honey coloured, are the main symptoms.

B. Individual Tumours of the Genital Tract

1. Cancer of the Uterine Cervix

Importance:

Carcinoma of the cervix is the most common of the genital cancer in the female, and the second most common malignancy in women. It arises in 1.6% of all women and (after allowing for cures), is the cause of death in 1%. In the community (U.S.A.) its incidence is some 44 per 100,000.

Aetiology:

The basic aetiology is unknown. Some associated characteristics, however, have been indentified.

a. *Coitus–*

Cervical cancer is very rare in nuns, and the incidence increases with early first intercourse, with marriage, with childbearing, and is especially high in prostitutes.

b. *Circumcision–*

Although cervical carcinoma is rare in women of the Jewish faith, circumcision is probably not the important factor, since other communities fail to show any relation to circumcision.

c. *Race* – Negresses are more susceptible, but social factors cannot be excluded.

d. *Social Standing* –

Carcinoma of the cervix is more common among lower social classes.

e. *Parity–*

The first pregnancy increases the risk ten-fold. Subsequent pregnancies increase this risk but minimally.

f. *Age–*

Cervical carcinoma is uncommon under the age of 40 years, but increases with age to occur maximally between 55 and 59 years.

All the above associations point to coitus as the major aetiologic factor, transmitting either a chemical or a viral agent. Recently work has shown that a virus, possibly herpes virus *11*, is closely associated with patients having preinvasive carcinoma of the cervix.

Pathology:

a. *Development*

There is probably a progression of cervical epithelium from the normal, through dysplastic epithelium and carcinoma-in-situ, to invasive carcinoma. This progression is irregular and may halt or even regress without treatment (Fig. 19.1). Although *all* invasive carcinomata probably arise from carcinoma-in-situ, it has been suggested that only the grosser forms of carcinoma-in-situ are likely to become, in time, invasive.

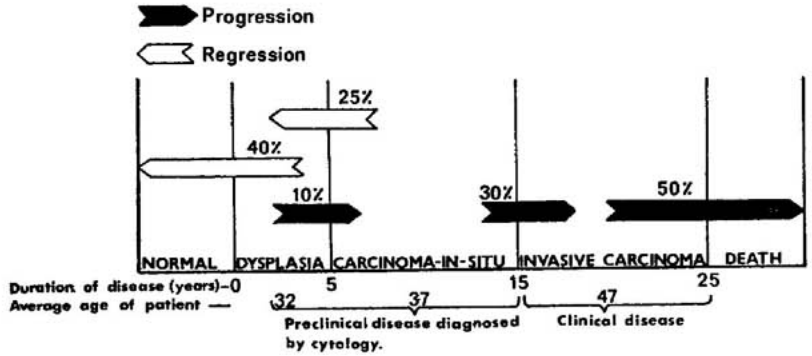

Fig. 19.1. Diagrammatic representation of the progression of cervical epithelium from normality to malignancy. (From Llewellyn-Jones, 1973).

When considering the earliest changes it has been suggested that during the metaplastic change of ectopic columnar epithelium on the cervix to stratified squamous epithelium, a certain number progress to a 'dysplastic' appearance. Dysplasia is characterized histologically by a hyperplasia of the deeper pricklecell layer and only a slight stratification of the layers.

In a small percentage of patients the dysplastic cervical epithelium may progress to carcinoma-in-situ (Bowen's disease of the cervix, or intraepithelial cancer). These terms imply that the epithelium has all the appearance of malignant tissue, but is not invasive. The diagnosis is suggested by abnormal cells seen in the cervical smear, but can only be confirmed histologically after a cone biopsy. Carcinoma-in-situ may progress to invasive cancer, or remain static for years.

Because carcinoma of the cervix may pass through a preinvasive stage, *cervical exfoliative cytology* has gained great popularity as a diagnostic tool in the detection of early cervical malignancy. This involves the removal of

cells from the squamocolumnar junction of the outer cervical os (Fig. 19.2) by the use of a wooden spatula. The cells so obtained are smeared on slides, fixed, stained, and examined for the appearance of normal, dysplastic, or malignant cells. The result may be graded into one of six categories. (Table 19.2)

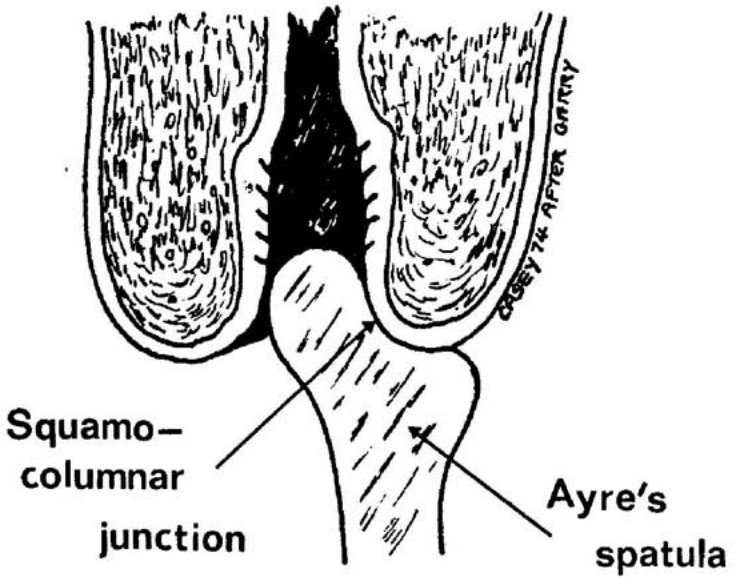

Fig. 19.2. Taking a cervical smear for cytology. The longer projection of Ayre's spatula is inserted into the cervical canal and rotated in a complete circle, scraping the squamo-columnar junction. (After Garrey *et al*, 1972.)

Cancer Cytology

1. No malignant cells seen.
2. Atypical cells not suggestive of malignancy.

2R. Atypical cells of doubtful significance *please repeat.*

3. Suspicious cells present.
4. Probable carcinoma cells present.
5. Carcinoma cells present.

Table 19.2. Grading of cervical cancer smears.

A cervical smear is a screening technique of high reliability, but it is not 100% reliable. Both false positive and false

negative reports will be obtained. A false positive result is one in which the smear shows abnormal or suspicious cells in the absence of cervical dysplasia, 'in situ' or invasive carcinoma. This figure will vary between one and ten per cent of cases, and the number will be reduced with more experienced cytologists. However, unless an adequate cervical biopsy is taken (which means "cone biopsy") a smear cannot to classed false positive.

A false negative smear report means that no abnormal cells are seen in a smear from a woman who has cervical dysplasia, "in situ" or invasive carcinoma. This may occur occasionally in a clinically obvious invasive tumour. It may occur if the tumour is of endocervical origin, or if it is on the ectocervix and not exfoliating cells. It also occurs when the technique fails to place abrasive cells on the slide for examination. The incidence of false negative smears is thought to be about 10%. For this reason a woman with a "normal" cervix should have a second smear a year after the first to ensure that the first report was not a false negative. Also a patient who has a clinically unhealthy cervix should have further investigation and often subsequent treatment, even if the first smear report is normal.

When a patient's cytology report is of grade 2R, 3 or 4, the smear should be repeated prior to further investigations. The second smear may either confirm the earlier cytological impression or show a normal smear. The patient with the normal second result should be followed up by regular smears. The patient with the persistently suspicious smear should now undergo a cone biopsy and a *fractional curettage* to exclude endometrial carcinoma contaminating the cervical smear. Here the endocervical and endometrial fragments are examined separately.

b. *Varieties of Cervical Malignancy*

Some 95% of cervical cancers are squamous cell carcinomas, and 5% are adenocarcinomas arising in the endocervix.

Carcinoma-In-Situ – (synonyms: Bowen's disease; intraepithelial carcinoma).

This term is used when the carcinoma is confined to the cervical epitheluim. It is a condition in which no clinical *symptoms* or *signs* are present, the condition being found usually by a cervical smear and confirmed by a cone biopsy. When the cone biopsy is performed the application of Schiller's Iodine to the cervix will reveal areas which do not stain (lack of glycogen in abnormal but not necessarily malignant cells). All such areas are suspicious and must be included in the biopsy.

If the cone biopsy shows the presence of carcinoma-in-situ, four factors must be considered in deciding treatment:

i. *The completeness of excision:* the pathologist may be able to state that the lesion appears completely excised or that the line of excision passes through the lesion.

ii. *Subsequent cytology:* a further smear should be taken 4-6 weeks later.

iii. *The woman's age.*

iv. *Her parity and desire for further children:* If excision has been inadequate, and cytology is still suspicious, the alternative treatment is either *a further biopsy*, or *hysterectomy after study of the vaginal vault* for further areas of carcinoma-in-situ. Generally, if the vault is normal, the uterus should be removed.

If excision is adequate and follow up cytology negative, treatment is more controversial. If the woman is young and desires children she should be managed by regular cervical smears. If she is over 40 and does not want more children her uterus should be removed; however, some authorities feel that cone biopsy is adequate if smears are taken regularly.

A positive smear from an apparently normal cervix during pregnancy is a difficult problem since cone biopsy may be complicated by abortion, premature labour or haemorrhage. Most commonly no interference will be attempted until after delivery.

Invasive Carcinoma of the Cervix

When the malignant epithelial cells of the cervix breach the basement membrane of the basal layer, carcinoma of the cervix is now "invasive".

Extension: Spread may be –

a. *Direct* –

This occurs first and involves the body of the uterus, the vaginal walls, the bladder, and the ligaments.

b. *Lymphatic* –

This usually follows but may precede direct spread. Node involvement is illustrated in Fig. 19.3.

c. *Haematogenous* –

Spread is rare and late.

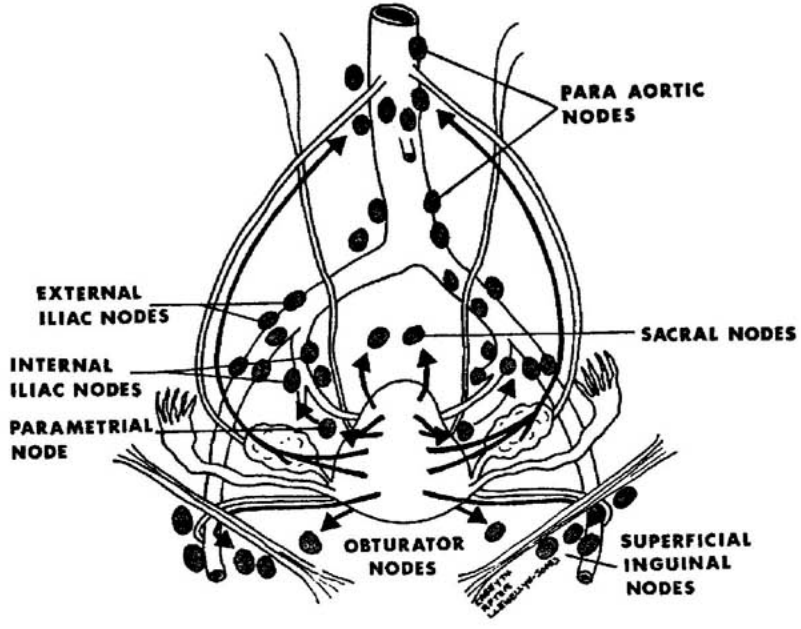

Fig. 19.3. Lymphatic drainage of the uterus. *Carcinoma of the cervix* most commonly involves the internal iliac and oburator nodes. Lympatic spread of *endometrial carcinoma* depends on the site of the lesion in the uterus, and is illustrated here by arrows. Fundal carcinoma for example, spreads to the external iliac nodes and sometimes to the superficial inguinal nodes. A growth nearer the cervix will behave as a cervical carcinoma. (From Llewellyn-Jones, 1973.)

Clinical Features:

Symptoms –

. *Irregular bleeding* – especially post-coital or post-menopausal.
. *Discharge* – due to infection a foul discharge may result.
. *Pain* – comes late and means that spread has reached the nerve trunks.
Cachexia – frequency, dysuria, rectal pain, and leakage of urine and faeces from the vagina are late symptoms.

Signs –

The cardinal signs are hardness, friability, fixation, and bleeding. The cervix may be normal looking, or mishapen. The cancer may be of an ulcerating type (endophytic), fungating type (exophytic), or of a nodular type.

Diagnosis:

Diagnostic procedures may include – A Schiller's iodine test; tissue biopsy; and fractional curettage.

Special procedures prior to treatment should include: An intravenous pyelogram, cystoscopy, sigmoidoscopy, barium enema, radiologic bone survey, and chest X-ray, to determine the extent of spread. Blood examination for haemoglobin and urea, and a urinary examination for blood, pus, and protein, are also done.

Classification:

Stage 0 – Carcinoma-in-situ.

Stage I – Carcinoma strictly confined to the cervix (extension to the corpus should be disregarded).

. *Stage Ia* – The cancer cannot be diagnosed by clinical examination.

Divided into:

i. Early stromal invasion,
ii. Occult cancer.

Another sub-group refers to the finding of a focus of carcinoma in the cervix of an excised uterus: — Stage Ia – post surgical.

. *Stage Ib* – All other cases of Stage I.

Stage II – The carcinoma extends beyond the cervix but has not extended to the pelvic wall. The carcinoma may involve the upper third of the vagina or extend into the parametrium.

. *Stage IIa* – No obvious parametrial involvement.

. *Stage IIb* – Obvious parametrial involvement.

Stage III – The carcinoma has extended on to the pelvic wall. On rectal examination there is no cancer – free space between the tumour and the pelvic wall or the tumour involves the lower third of the vagina.

Stage IV – The carcinoma has extened beyond the true pelvis or has involved the mucosa of the bladder or rectum. Bullous oedema as such does not permit allotment of a case to Stage IV.

Treatment:

Stage 0 – Discussed previously.

Stage Ia – Radical hysterectomy (Rutledge, Class I or II) and pelvic lymphadenectomy.

Rutledge Class I – for micro-invasion and questionable superficial invasion. In this technique the ureters are deflected laterally without dissection from the ureteral bed and the parametrium is clamped wider than usual but *medial* to the ureter. A good vaginal cuff is also taken.
Rutledge Class – II for superficial invasion and limited invasion. Here the ureters are mobilised and the uterine vessels are divided *medial* to the ureters (so preserving their blood supply). The uterosacral ligaments are divided low down and the upper third of vagina is excised. The technique removes more paracervical tissue and vagina.

Stage Ib and early well differentiated Stage II

– These cases do equally well with radical surgery

or radical radiotherapy. The decision is made by the gynaecologist and radiotherapist in consultation. If surgery is elected then a Rutledge Class III hysterectomy and pelvic lymphadenectomy is preferred.

Rutledge Class III – The uterine artery is ligated at its origin. The ureters are well mobilised and the uterosacral ligaments divided near the rectum. Half of the vagina is excised.

If radiotherapy is decided upon post-irradiation biopsies are preferred at 4 and 6 weeks. Combined surgical and radiotherapeutic treatment is not recommended.

Stage II Advanced (or anaplastic) and Stage III

– External irradiation with biopsy and assessment at 4 and 6 weeks. If an unfavourable response to radiotherapy, radical surgery to be considered which may involve exenteration. If a favourable response is seen radium and further external radiotherapy is carried out.

Stage IV – Radiotherapy and/or radical surgery.

N.B.: The term "Wertheim's hysterectomy" should *NOT* be used as it is misleading. Wertheim described a radical hysterectomy which did *NOT* include a pelvic lymphadenectomy.

Results of Treatment:

	% 5 year survival	
Stage 0	100	
Stage I	90	
Stage II – a	70)	
Stage II – b	60)	– 68
Stage III – a	45)	
Stage III – b	30)	– 35
Stage IV	15	
All stages	50	

Complication and Causes of Death:

An obstructed cervical canal predisposes to the development of a *pyometra*. Direct spread may cause the formation of *fistulae*, including vesico-vaginal, vesico-cervical, and recto-vaginal fistulae. *Ureteric obstruction* may lead to the development of hydronephrosis, pyonephrosis, and renal failure with uraemia.

Uraemia is the cause of death in more than 50% of cases. Other causes include cachexia, haemorrhage, intestinal obstruction, peritonitis, and secondary growths.

2. Cancer of the Body of the Uterus

The ratio of endometrial to cervical carcinoma is about 1:2 in white women. It is usually a disease of post-menopausal women and less than 25% of endometrial carcinoma occurs in menstruating women. The average age of detection is 57 years. Sarcoma of the uterus is rare and will not be discussed here.

Aetiology:

Endometrial carcinoma is found more commonly in women who have had:

a. A late menopause,
b. Prolonged heavy menses late in their menstruating history
c. Suffered from obesity, hypertension or diabetes,
d. A poor fertility index.

Other associated factors are –

Prolonged oestrogen influence or high levels of oestrogens from an endogenous source. The result is endometrial hyperplasia with the production of cystic glands, hypertrophy of cells lining the glands, and hyperplasia of the stroma. This hyperplasia can become so excessive that the glands lose their normal orderly pattern, mitoses increase in number, and it becomes difficult to distinguish from true early carcinoma.

High oestrogen levels may occur with –

i. Oestrogen therapy at menopause.
ii. Late menopause – an ovulation is common at the end of menstrual life, and unopposed oestrogen activity can be excessive.
iii. Feminising ovarian tumours.
iv. Ovarian stromal hyperplasia.

Pathology:

The *macroscopic* appearance is illustrated in Fig. 19.4.

The *histological* appearance is that of a departure from the normal pattern. The glands are disorderly and atypical in outline and the cells show increased activity and mitoses.

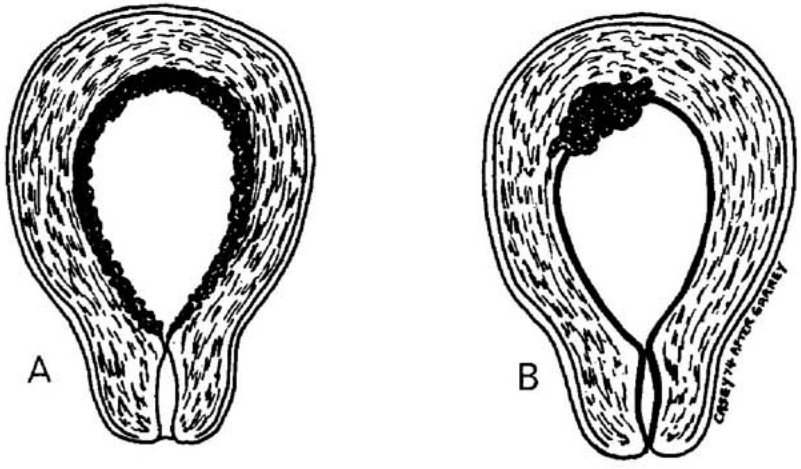

Fig. 19.4. Macroscopic types of endometrial carcinoma.
A. *Diffuse type* – a large surface of the endometrium is involved. The appearance is of a thickened polypoid lining with areas of necrosis and ulceration. It may grow through the myometrium or extend down the cervical canal.
B. *Localised type* – only a small area of the endometrium appears to be involved. The lesion may be small ulcer or a polypoid growth.

Spread:

a. *Direct* spread is slow and progresses into the myometrium and down into the cervix.

b. *Lymphatic* spread tends to be late in onset and is illustrated in Fig. 19.3.

c. *Haematogenous* spread is rare and occurs terminally in liver, lungs, and bones.

Clinical Features:

Irregular bleeding peri-and post-menopausally is the only constant symptom. Consequently, any such bleeding should always be investigated by a diagnostic curettage. Pain or discharge from an ulcerated growth may also be present. On bimanual examination the uterus is small with the early lesions, but may be enlarged by a pyohaematometra. In advanced cases the uterus may be fixed.

Diagnosis:

On history, bleeding in post-menopausal women must be considered due to carcinoma until proven otherwise.

A *fractional curettage* is carried out and the fragments examined histologically.

If carcinoma is diagnosed a pre-treatment work-up, similar to that described for cervical cancer, is carried out to help stage the malignancy.

Classification of Carcinoma of the Corpus Uteri

Stage 0 – Carcinoma-in-situ. Histological findings are severe atypical hyperplasia which is suspicious of malignancy.

Stage I – The carcinoma is confined to the corpus.

Stage Ia – The length of the uterine cavity is 8 cm. or less.

Stage Ib – The length of the uterine cavity is more than 8 cm.

The Stage I cases should be sub-grouped with regard to the histological type of adenocarcinoma as follows:

G1 – highly differentiated adenomatous carcinomas.

G2 – differentiated adenomatous carcinomas with partly solid areas.

G3 – predominantly solid or entirely undifferentiated carcinomas.

Stage II The carcinoma has involved the corpus and the cervix extending to the serosal surface or into the parametrial tissue.

Stage III – The carcinoma has extended outside the uterus to involve the parametrium to the pelvic walls but not outside the true pelvis.

Stage IV – The carcinoma has extended outside the true pelvis or has obviously involved the mucosa of the bladder or rectum. Bullous oedema as such does not permit allotment of a case to Stage IV.

Treatment:

Stage 0 – Extrafascial total hysterectomy and bilateral salpingoophorectomy (THBSO) with cuff of vagina.

Stage I – Preoperative radium followed by surgery. The use of preoperative radium reduces the incidence of vault recurrences.

Consideration may be given to radium packing of the uterus as an alternative management in poor risk patients.

Surgery in well-differentiated tumour means extrafascial THBSO with cuff of vaginal.

In the case of anaplastic tumours a radical hysterectomy (Rutledge Class II) and pelvic lymphadenectomy is indicated.

Surgery should be carried out within 3 days of removal of the radium or after 6 weeks.

Stage II – Treat as for carcinoma of cervix where spread to cervix is proven by wedge biopsy.

Stage III – Diagnostic laparotomy, external irradiation and progesterone therapy to be carried out prior to or after commencement of treatment.

Stage IV – Radiotherapy and/or progesterone therapy.

Results of Treatments:

	% 5 year survival
Stage 0	100
Stage I	90
Stage II	60
Stage III	40
Stage IV	25

The prognosis largely depends on the stage of the disease and the histological grade.

3. Ovarian Tumours

Cancer of the ovary is the most common *cause of death* in women from cancer of the genital tract. Next in importance is the cervix, followed by the uterus. The higher death rate is largely the result of *late diagnosis* because ovarian cancer is so often symptomless until the disease is well advanced.

Because ovarian malignancy often has counterparts in the benign tumours of the ovary these will be considered here.

Classification: *Tumours*	*Proportional Incidemce %*
Benign – usually cystic	
Functional cysts (follicular; corpus luteum)	24
Serous cystadenoma	20
Pseudomucinous cystadenoma	20
Teratoma (benign)	20
Endometrial cyst	5
Fibroma	5
Malignant	
Primary: Cystadenocarcinoma– a. Pseudomucinous b. Serous	5

Rare Tumours – usually solid		*Proportional Incidence %*
Feminizing:	Granulosa-cell tumour Thecoma	
Virilizing:	Arrhenoblastoma	1
Neuter:	Disgerminoma	
Teratoma (malignant)		
Secondary:	from breast, stomach, opposite ovary, colon, uterus.	20% of ovarian malignancy.

The above classfication is a gross oversimplification of the wide variety of tumours found in the ovary. Because the ovary consists of sex cells that are totipotential and of multipotential

mesenchymal cells, almost any tumour may result, and hundreds of various types have been recorded.

Benign Ovarian Tumours

a. *Functional Cysts* are included under the above heading only because they are a very common benign enlargement of the ovaries. Functional cysts are not new growths.

   Follicular cysts arise from distension of a Graafian follicle and are seen typically in metropathia haemorrhagica, and often in a normal woman. They arise as a result of a degeneration of an ovum and a persistence of granulosa or theca cells which remain functional. Follicular cysts as a rule are never more than 5 cm. in diameter.

   Most follicular cysts are asymptomatic but some may cause menstrual irregularities (due to oestrogen production the extreme situation being metropathia haemorrhagica. In the majority of cases follicular cysts are self curative and no interference, except perhaps puncturing, should be attempted if a cyst is discovered accidentaly at laparotomy.

   Corpus Luteum cysts are seen in pregnancy and sometimes follow on the corpus luteum of menstruation. They may cause short periods of amenorrhoea followed by prolonged uterine bleeding. Ordinarily corpus luteum cysts regress spontaneously and need no surgical interference.

b. *Serous Cystadenoma* is a unilocular or multilocular cyst lined by columnar epithelium containing a thin, watery, colourless secretion. It is bilateral in 1/3 cases, and may reach 10 cm. or more in diameter.
   Serous cystadenomas occur commonly in the 20 to 50 years age-bracket, and their importance lies in the potential malignant change that can occur. This risk is especially high (about 30%) in the cystadenoma with intracystic papillary projections. Histological grading of malignancy is difficult and may be misleading.

c. *Pseudomucinous Cystadenoma* is a very common ovarian tumour. It consists of a collection of cysts lined by columnar epithelium filled with a mucinous substance which may be colourless, green or brown, depending on the presence of blood pigments.

   This tumour may grow to a very large size (up to 50 cm. diameter) and may be bilateral in about 10% of cases but only rarely becomes malignant.

   Rarely, when the contents of a mucinous cyst are spilled into the peritoneal cavity, a complication termed *pseudomyxoma peritonei* may ensue. The epithelial cells of the tumour seed the peritoneal cavity and secrete semi-solid psuedo-mucinous material which tends to fill the abdomen. Patients slowly become cachexic, require laparotomies to remove the material, and ultimately succumb.

d. *Teratoma (Dermoid Cyst)*

   The benign teratoma may occur at any age the peak being between 20 and 40 years of age. It is bilateral in some 10% of cases. The cyst is usually unilocular and often contains hair, pultaceous material from sebaceous glands, and teeth or cartillage. A special form of the teratoma may contain thyroid tissue and is termed struma ovarii.

e. *Endometrial cysts* (Chocolate cysts) of the ovary are a cause of ovarian enlargement and are discussed under *Endometriosis* in Chapter 15.

f. *Fibroma*

   This is a tumour of the ovarian connective tissue and is a solid, hard, white growth. It occurs most commonly in middle age. Although it contains no muscle elements it behaves like the uterine fibroid being subject to the same complications. The fibroma may cause ascites (20% of cases), and when associated with a hydrothorax the combination is termed Meigs Syndrome.

Complications:

a. *Torsion* of the pedicle is the most common complication, and unless treated gangrene will occur. Symptoms begin some 4-5 days before the patient is seen and include pain in the illiac fossa, nausea and vomiting. Signs are of a pelvic mass with localised tenderness and peritonism. Laparotomy is usually required.

b. *Haemorrhage* into or from a cyst may cause pain or tenderness that is self-limiting.

c. *Rupture* of a cyst. Rupture of a small follicular cyst gives rise, in many women, to pain at the time of ovulation, termed "mittelschmertz" (middle pain). Rupture of a large ovarian cyst may bring on a sudden acute abdominal pain, sometimes with vomiting. Tenderness, guarding, rigidity, and shoulder tip pain follow. Fluid may be demonstrated in the abdominal cavity.

d. *Degeneration* of fibromas is similar to that of uterine fibroids.

e. *Infection* is a rare complication and may follow torsion of a cyst.

f. *Malignant* change may complicate the cystadenomas.

Malignant Ovarian Tumours

Primary cancers of the ovary are mostly malignant counterparts of the serous and pseudomucinous cystadenomas. The three main types of pathology include the serous cystadenocarcinoma (40% bilateral), and the pseudomucinous cystadenocarcinoma (18% bilateral), and the anaplastic carcinomas (about 50% bilateral), most of which are serous in origin. Pure adenocarcinomas resembling endometrial cancer also occur. The remaining malignancies are made up by the rare tumours listed in the classification.

Clinical Features of Ovarian Tumours:

Age: Ovarian tumours may occur at any age but are more commonly found in the 30-60 age group.

Symptoms:

Ovarian tumours give rise to (mechanical) symptoms late in their development, so that on admission to hospital about 50% of the patients already have clinically recognizable metastases. First symptoms are often abdominal enlargement, chronic low abdominal pain, vaginal bleeding, nausea and vomiting, weight loss, and bowel and urinary disturbances.

All ovarian tumours may secrete some oestrogen. This is because the growing tumour stimulates the production of granulosa/lutein-like cells from the surrounding ovarian stroma. As a consequence, at least 30-50% of post-menopausa

women with an ovarian tumour (benign or malignant) may have evidence of oestrogen production and, therefore, often present with bleeding due to endometrial hyperplasia and subsequent breakdown.

Signs:

Abdominal swelling and a palpable tumour, with ovarian enlargement on bimanual examination, help to deterimne whether the tumour is from the ovary. Assess whether the swelling is attached to or part of the uterus, whether it moves with the cervix, and whether the uterus or ovary is independent of it. Pointers in favour of malignancy include ascites, fixed nodules in the pouch of Douglas, history of fast enlargement, solid, bilateral swelling, oedema of a leg, and evidence of distant metastases.

Differential Diagnosis:

In diagnosing any ovarian enlargement one has to distinguish from:

- distended bladder
- pregnancy
- obesity
- uterine fibroids
- ascites
- appendiceal abscess
- ectopic pregnancy
- hydrosalpinx
- pseudocyesis
- ectopic kidney
- tumours of the colon

Diagnosis:

There is no satisfactory method of ascertaining malignancy apart from laparotomy and biopsy. Prior to this, however, a full diagnostic work-up is carried out including upper and lower gastro-intestinal investigation to rule out metastases from a primary gastro-intestinal tumour. Breast examination, intravenous pyelogram, search for skeletal metastases, and paracentesis and cytology are also carried out.

At laparotomy the following characteristics will suggest malignancy:

. A solid tumour.
. Growth of tumour through its capsule.
. Haemorrhage visible through the wall.
. Large blood vessels on the surface.

. Bilateral tumour.
. Blood-stained ascitic fluid.
. Metastatic deposits on the peritoneum or omentum.
. Adhesion of tumour to other structures.

After diagnosing the ovarian tumour treatment for the appropriate stage should be carried out.

Classification and Treatment:

Stage I – growth limited to the ovaries;
Stage Ia growth limited to one ovary, no ascites;
Stage Ib growth limited to both ovaries, no ascites;
Stage Ic growth limited to both ovaries, ascites present with malignant cells in the fluid.

Surgery followed by Chlorambucil therapy. Chlorambucil in a dosage of 30 mgm I.V.I. is repeated after one week if platelets and W.B.C. are satisfactory and then followed up with chlorambucil by mouth for 3 months, a break of three months and then a further three months of Chlorambucil.

Further chemotherapy is given after twelve months only if some special indication is present.

Stage II - growth involving one or both ovaries with pelvic extension

Treatment as for Stage I with radiotherapy if there is localised area of tumour remaining in the pelvis.

Stage III - growth involving one or both ovaries with widespread intra-peritoneal metastases to the abdomen

Biopsy of the tumour, followed by radiotherapy, exploratory laparotomy and follow-up chemotherapy.

Stage IV - growth involving one or both ovaries with distant metastases outside the peritoneal cavity

Palliative therapy.

Results:

The most important factors in prognosis are the anatomic stage and the histologic type and grade of neoplasm.

Stage	*5 year survival %*
I	75
II	65
III	45
IV	10
Overall	30

The above figures look satisfactory at a glance, but it should be pointed out that the bulk of the patients fall into Stage IV where survival is very short.

The most common cause of death in ovarian malignancy is intestinal obstruction.

4. **Chronic Epithethial Abnormalities of the Vulva:**

A dystrophy is basically a disorder arising from defective or faulty nutrition. The epithelium of the vulva, so much influenced by the sex hormones, is especially prone to changes which may range from atrophy to premalignancy.

a. *Senile atrophy* of the vulva is a physiological change attendent on hormonal withdrawal at menopause. It consists of atrophy and shrinkage of the labia with contracture of the introitus. It is symptomless apart from the possibility of dyspareunia.

b. *Kraurosis vulvae* is characterized by reduction of the vulval skin keratin so that the corium tends to be exposed, bright red, and susceptible to infection. In time sclerosis and atrophy lead to shrinkage and contracture of the introitus. The condition is most common after menopause but may occur in younger women and symptoms may include dyspareunia, pruritus, and tenderness. Risk of malignancy is very slight.

c. *Leukoplakia vulvae* is characterized by a thick, hard white skin which cracks easily. It differs from kraurosis in that the vulval skin has increased keratin layer. The affected areas are usually the labia, clitoris, and perineum, but not the vestibule or vagina. Leukoplakia is usually post-menopausal and the main symptom is intractable pruritus. Scratching may lead to ulceration and infection. Importance of leukoplakia lies in its tendency to progress to carcinoma, and biopsy should be carried out. Steroid creams give best relief from symptoms.

d. *Lichen sclerosus* probably includes a collection of dermatological conditions of the vulva. It may occur at any age and may affect other parts of the body. It is symmetrical, affecting the flexor surfaces as an eruption of round, flat, violet papules that are intensely pruritic (lichen planus). Constant scratching, infection, and trauma may produce an end-stage of white patches and contractures that are difficult to differentiate from leukoplakia (lichen sclerosus). Lichen simplex, also known as neurodermatitis, occurs in the vulval skin. The condition is secondary to scratching probably as a result of a pruritus of psychological aetiology.

e. *Pruritius Vulvae*

The causes of pruritus may be divided into general and local causes. These will be simply listed here.

Local Causes:

Vaginal discharges (Common) –
. Trichomonas,
. Monilial.

Vulval epithelial disorder –
. Leukoplakia,
. Lichen planus and other dermatological disorders,
. Primary cancer.

Allergic dermatitis.

General Causes:

Diabetes Mellitus,
Jaundice,
Psychological causes (common),
Medication, e.g. cortisone or antibiotics,
General sensitivity reactions.

5. Vulval Cancer

This is a rare malignancy comprising some 3% of all genital cancers, and it occurs mostly in women over the age of 60 years. The invasive stage may be preceded by an intraepithelial stage (Bowen's disease). The associated or preceding vulval lesions are leukoplakia, and less commonly kraurosis, diabetic vulvitis, radiation vulvitis, and venereal disease.

Clinically the presenting complaints may include pruritus, discharge, bleeding, pain, the presence of a tumour, or an ulcer.

Some 95% of cases are squamous cell carcinomas which involve usually the labium majus, labium minus, or the clitoris. Vulval carcinoma spreads locally to the vagina, urethra, and anus, and by lymphatics to the superficial inguinal, femoral, and external iliac nodes. Deep node involvement indicates a poor prognosis.

Treatment consists of radical surgery including complete vulvectomy and pelvic lymph node resection. With early lesions (less than 3 cm diameter) the 5 year survival is about 7%. Radiotherapy has no place in treatment since the vulval skin is extremely sensitive to radiation.

6. Vaginal Malignancy

Primary carcinoma of the vagina is less common than secondary vaginal malignancy (especially cervical, uterine, breast, bowel, bladder, or ovary). The tumour is usually located high up on the posterior vaginal wall and may give rise to symptoms of bleeding, contact bleeding, discharge, pain, and urinary symptoms with bladder involvement.

In diagnosing the primary vaginal carcinoma metastic malignancy, endometriosis, and granulamatous lesions are excluded and a biopsy is performed.

Intracavity radiotherapy in conjunction with external pelvic irradiation is the treatment of choice. Extensive surgery may be considered in some cases. When the tumour is confined to the vaginal wall the 5 year survival is over 50%.

7. Hydatidiform Mole and Choriocarcinoma

These are rare toumurs which usually arise in the placenta. Rarely a choriocarcinoma may arise in a teratoma.

Hydatidiform Mole:

Incidence:

The incidence of hydatidiform mole varies between geographical regions. In the U.S.A. it occurs in 1 out of 2,000 deliveries; Australia 1 out of 800 deliveries; in Hong Kong 1 out of 600 deliveries.

Age and Parity:

Average age of incidence is about 30 years, occurring solely during the reproductive years.

Pathology:

This condition shows a hydropic swelling and formation of vesicles in the placental villi, with a relative lack of blood vessels. In the gross specimen the appearance may simulate a bunch of white grapes (5-10 mm. diameter).

Even the benign moles can invade the uterine wall causing massive haemorrhage during removal of the tumour. The invasive nature of this tumour may also give rise to embolisation to other organs, but this does not necessarily indicate malignancy.

Clinical Features:

. *Uterine Bleeding* is the most common presenting symptom and usually occurs around 12 weeks of amenorrhoea. Bleeding may be profuse or slight.

. *Excessive uterine enlargement* is seen in about 50% of cases, while 25% have normal and 25% smaller uterine sizes.

. *Pre-eclampsia* before the 18th week of pregnancy may occur. Absence of foetal parts and a "snow storm" appearance is diagnostic on utltrasonic echoscopy.

. *Nausea and Vomiting* are more common and may be excessive.
. In addition to the above, there may be *uterine colic* due to attempts at expulsion, and enlarged luteal cysts may be palpated in the ovaries.

Diagnosis:

The clinical history and examination may be supplemented by ultrasonic echoscopy. Additional evidence may be gained from measuring the titres of H.C.G. Titres greater than 100,000 international units, especially after day 100, is suggestive of hydatidiform mole.

A plain X-ray will show no calcification of the foetus.

Treatment:

Hydatidiform mole is generally treated by evacuating the uterus using a sponge, forceps, and a blunt curette. A syntocinon drip is usually kept running to obtain good uterine contractions and to assist in avoiding perforation of the uterus. Follow-up of patients who have had a hydatidiform mole includes a weekly check on the chorionic gonadotrophin level for one month, then monthly checks for 3 months and, finally, three-monthly checks for a further year. A positive U.C.G. result may indicate persistence of molar tissue but a pregnancy should be excluded before performing any further curettes. A persistent positive U.C.G. test following apparent removal of a mole may indicate invasion or metastases and so methotrexate may be given. A hysterectomy is rarely performed for a hydatidiform mole in our present management.

Choriocarcinoma

Probably 2-5% of hydatidiform moles become malignant, whilst some 50% of choriocarcinoma arises in a hydatidiform mole.

The tumour is anaplastic and very invasive almost always producing metastases in distant organs or tissue. The metastases are widespread, usually to the lung, vagina, and brain.

Diagnosis:

The diagnosis is suspected from the clinical history of a previous hydatidiform mole, and reappearance of haemorrhage. An increase in the titre of U.C.G. along with histological examination completes the diagnosis. After an abortion, term pregnancy, or even ectopic gestation, repeated or continual bleeding is usually due to retained tissue. A curettage is indicated if bleeding perisists. If bleeding recurs and is accompanied by rising H.C.G. titres the diagnosis is possible choriocarcinoma until proven otherwise.

Treatment:

Because metastases occur so early surgical treatment alone is disappointing as is radiotherapy. Methotrexate has been used very successfully to treat even widespread metastatic disease, with 5 year survival rates of some 60%.

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Adverse Reactions

During studies 'CENDEVAX' was shown to be highly attentuated and well tolerated – clinical reactions attributable to 'CENDEVAX' have been infrequent and mild. Slight swelling of lymph nodes in the regions of the head and neck has been reported in approximately 9% of susceptible vaccines. Rarely, slight temperature elevation and mild rash have occurred. In general, reactions have been reported to occur within one to four weeks post-vaccination; all have been transient and without sequelae. Allergic reactions (those seen within 72 hours) have very rarely occured.

The occurrence of arthralgia and, more rarely, arthritis, has been observed chiefly in adult women following vaccination with rubella vaccines. With the 'Cendehill' strain these reactions occurred only infrequently and were mild and of short duration.

Contra-indications and Precautions

Rubella Virus Vaccine, live, attenuated, is strictly contra-indicated in pregnant women since the effect of the attenuated virus on the foetus is not known. Following vaccination adequate measures must be taken to insure against conception for at least 2 menstrual cycles. As for every immunizing agent. 'CENDEVAX' is contra-indicated in the presence of acute febrile illness or chronic debilitating disease, and in subjects under corticosteroid therapy. Known hypersensitivity to rabbits and neomycin is also a contra-indication.

It is recommended that rubella vaccination be given at least one month before or after administration of other live virus vaccinations. Vaccination should be delayed until at least six weeks after gamma globulin therapy or blood transfusion because of the possible suppressive effect of passive antibodies.

'CENDEVAX' should not be administered to infants less than one year old because of possible interference from persisting maternal rubella antibodies.

Excretion of the live attenuated rubella virus from the throat has occurred in the majority of susceptible individuals administered the vaccine. There is no definitive evidence to indicate that such virus is contagious to susceptible persons who are in contact with the vaccinated individuals. Consequently, transmission, while accepted as a theoretical possibility, has not been regarded as a significant risk by expert groups in the recommendations for the use of the vaccine. The lyophilized vaccine should be stored at low temperatures 2° to 8°C (35° to 46°F) and protected from light. Exposure to freezing temperatures will not harm the lyophilized vaccine but may damage the diluent vial. The vaccine should be administered promptly, after reconstitution by the subcutaneous route only. When necessary, the reconstituted vaccine may be held at a temperature not higher than 8°C (46°F) for not more than 8 hours prior to use.

Presentation

'CENDEVAX' ('Cendehill' strain) is available as single dose units, each unit consisting of a single dose vial of lyophilized vaccine, a cartrigde containing 0.5 ml sterile diluent and a disposable syringe fitted with a 25 gauge $\frac{5}{8}$" needle.

'Fefol' 2 'Spansule' Capsules

Composition

Each 'FEFOL' 2 'SPANSULE' capsule contains 270 mg exsiccated ferrous sulphate and 300 mcg folic acid.

The release pattern of 'FEFOL' 2 'SPANSULE' capsules differs significantly from that of other 'Spansule' Capsules. The ferrous sulphate pellets are specifically coated so that release is delayed and timed to occur mostly in the upper part of the small intestine, minimizing the risk of gastrointestinal upset.

Actions

'FEFOL' 2 'SPANSULE' Capsules provide the body with ferrous sulphate – iron in its most efficient (bivalent) form. Ferrous sulphate acts to correct simple iron deficiency and iron deficiency anaemia by providing the iron necessary for the production of haemoglobin, certain iron-containing enzymes and compounds which store iron for the body's future needs.

'FEFOL' 2 'SPANSULE' Capsules contain sufficient folic acid to prevent the development of folate deficiency during pregnancy.

Indications

Prophylaxis of iron and folic acid deficiency in pregnancy.

Dosage

One 'FEFOL' 2 'SPANSULE' capsule a day throughout pregnancy, or as directed.

Adverse Reactions

Nausea and other alimentary symptoms usually encountered during iron therapy are unlikely to occur with 'FEFOL' 2.

Precautions

The folic acid content of 'FEFOL' 2 is adequate for prophylaxis during pregnancy and is unlikely to mask pernicious anaemia should this condition be present.

Overdosage

Symptoms – As 'FESPAN SPANSULE' Capsule.

Treatment – Includes gastric lavage, cathartics (if corrosion is not far advanced), enema and the administration of demulcents. Measures to combat shock, dehydration and respiratory failure may be necessary. Give water, milk or "universal antidote" to delay absorption of ingested iron. Then remove by gastric lavage or emesis. Wash out stomach with sodium bicarbonate solution (50g sodium bicarbonate to a litre of water or 3-4 tablespoonfuls per pint) or with milk. The sodium bicarbonate forms a poorly soluble, and therefore less toxic, ferrous carbonate. Leave 250 ml (approximately a cupful) of sodium bicarbonate solution or the same quantity of milk in the stomach. To relieve irritation, 250 ml of milk may be given alternatively with 5 g bismuth subcarbonate every hour. Desferrioxamine mesylate has been recommended for the treatment of acute oral iron poisoning .The recommended dosage is 8 g by nasogastric tube followed by parenteral procedures for mobilisation of iron from storage sites. As this compound is not without toxic effects, the reference cited should be carefully read before administration. (Reference: Goodman & Gilman, The Pharmacological Basis of Therapeutics, 4th Ed., page 954).

Presentation

'FEFOL' 2 'SPANSULE' Capsules are available in containers of 30 and 150.

N.H.S. AVAILABILITY
General: Capsules 30, 2 repeats.

'FESPAN SPANSULE' CAPSULE

Composition

Each 'FESPAN SPANSULE' capsule contains 320 mg of dried ferrous sulphate B.P., equivalent to 100 mg of elemental iron.

The release pattern of 'FESPAN SPANSULE' capsules differs significantly from that of other 'Spansule' capsules. The ferrous sulphate pellets are specifically coated so that release is timed to occur mostly in the upper part of the small intestine, minimizing the risk of g.i. upset.

Actions

'FESPAN SPANSULE' capsules provide the body with ferrous sulphate – iron in its most efficient (bivalent) form. Ferrous sulphate acts to correct simple iron deficiency and iron deficiency anaemia by providing the iron necessary for the production of haemoglobin, certain iron-containing enzymes and compounds which store iron for the body's future needs.

Indications

For use in simple iron deficiency or iron deficiency anaemia where oral iron is indicated. Since risk of gastrointestinal upset is minimal, 'FESPAN SPANSULE' capsules are of particular value in patients intolerant to conventional iron and those prone to gastrointestinal upset.

Dosage and Administration

Adults and Children – One 'FESPAN' capsule daily. Children too young to swallow the capsule can be given the contents in a spoonful of soft, cool food.

Adverse Reactions

Because of the design of the product, gastrointestinal discomfort occurs rarely.

Overdosage

Symptoms: Symptoms of massive overdosage include lethargy, vomiting, diarrhoea, weak and rapid pulse and low blood pressure. Local corrosion of the stomach and small intestine may result from massive overdoses of iron by mouth, and sufficient iron may then be absorbed through the injured mucosa to produce systemic damage. Shock is commonly present. Stools are black and tarry. Bronchial pneumonia may be a complication.

Treatment: Includes gastric lavage, cathartics (if corrosion is not far advanced), enema and the administration of demulcents. Measures to combat shock, dehydration and respiratory failure may be necessary. Give water, milk or "universal antidote" to delay absorption of ingested iron. Then remove by gastric lavage or emesis. Wash out stomach with

sodium bicarbonate solution (50 g sodium bicarbonate to a litre of water or 3-4 tablespoons per pint) or with milk. The sodium bicarbonate forms a poorly soluble, and therefore less toxic, ferrous carbonate. Leave 250 ml (approximately a cupful) of sodium bicarbonate solution or the same quantity of milk in the stomach. To relieve irritation, 250 ml of milk may be given alternately with 5 g bismuth subcarbonate every hour. Desferrioxamine mesylate has been recommended for the treatment of acute oral iron poisoning. The recommended dosage is 8 g by nasogastric tube followed by parenteral procedures for mobilisation of iron from storage sites. As this compound is not without toxic effects, the reference cited should be carefully read before administration. (Reference: Goodman & Gilman. *The Pharmacological Basis of Therapeutics*, 4th Ed., page 954).

Presentation

'FESPAN SPANSULE' capsules are available in containers of 30 and 150.

N.H.S. AVAILABILITY
General: Capsules, 30, 2 repeats.

'Furadantin'

Composition

Each 'FURADANTIN' tablet contains nitrofurantoin 50 mg or 100 mg. Each ml of 'FURADANTIN' suspension contains nitrofurantoin 5 mg.

Indications

'FURADANTIN' is indicated in infections of the urinary tract and associated organs: cystitis, pyelitis, pyelonephritis, urethritis, infection before and after prostatectomy, infection after transplantation of ureters, infection after passage of instruments.

Dosage

'FURADANTIN' Tablet Dosage
Acute Infections – For acute uncomplicated urinary tract infections except acute prostatitis and pyelonephritis the average adult dose is one 50 mg tablet 4 times daily. If improvement does not occur within 2-3 days increase this dosage to a maximum of 400 mg daily. For acute prostatitis and pyelonephritis the average adult dose is 100 mg 4 times daily.

Continue administration for at least 3 days after sterility of the urine is attained.

Continued infection suggests the need for re-evaluation of antibacterial sensitivity of organism and thorough study of the urinary tract.

If nausea occurs reduce the dosage.
Chronic or Recurrent Infections – The average adult dose is 400 mg daily. This is given with meals and with food or milk on retiring. More exact dosage is 5-7 mg/kg (2.2-3.2 mg/lb) body weight /24 hours, not to exceed 400 mg per day.

'FURADANTIN' Tablets
Average Dosage Chart

Body Weight		Average Dose with each meal and at bedtime			
lb	kg	5 mg/kg		7 mg/kg	
		No. Tab.	Size Tab.	No. Tab.	Size Tab.
60— 84	27—38			1	50 mg
85—114	39—51	1	50 mg	1½	50 mg
115—139	52—63	1½	50 mg	1	100 mg
140—169	64—76	1	100 mg	1	100 mg

If therapy is indicated beyond 10-14 days, consideration should be given to reducing dosage to a ¼ or ½ the full therapeutic dose. In chronic prostatitis, as an adjunct in treatment, the average dose is four 100 mg tablets daily for 10-14 days. Depending on response to therapy, the dosage may then be reduced to 100 to 200 mg daily. This reduced dosage should be followed for a period of 1 to 3 months depending upon the clinical and laboratory response.

'FURADANTIN' Suspension Dosage.
Adults: 2-4 five ml doses 4 times daily with meals, and with food or milk on retiring. Children: ½ to 3, five ml doses 4 times daily with food or milk.

Under 15 pounds of body weight, the dose should be determined on the basis of 5 to 7 mg per kg (2.2—3.2 mg/lb) per 24hours. 'FURADANTIN' should not be administered to infants under one month of age.

NOTE—

1. An adequate dosage level should be maintained constantly for at least 5-7 days, and for 3 days after the urine is sterile.

'FURADANTIN' Suspension
Average Dosage Chart

Body Weight		No. of 5ml doses 4 times daily	
lb	kg	5 mg/kg	7 mg/kg
15— 24	7—10		1/2
25— 49	11—22		1
50— 84	23—38		2
85—114	39—51	2	3
115—139	52—63	3	4
140—169	64—76	4	4

2. For long-term continuous therapy, consideration should be given to finding the lowest effective dose. This is especially important in elderly patients.
3. Unlike the sulphonamides, 'FURADANTIN' does not call for increased fluid intake; forcing fluids beyond normal merely dilutes the antibacterial concentration of the urine.
4. Some of the break-down metabolites frequently cause a yellowish-brown discolouration of the urine which is entirely harmless, The Hay's test may occasionally give a false positive.

Adverse Reactions

There are few adverse reactions to 'FURADANTIN' therapy. Nausea may occur, but is greatly reduced by careful adjustment of dosage, by showing the rate of administration and by taking the drug with food or milk. Should the nausea persist, the drug should be withdrawn.

Occasional skin rashes, which have disappeared on discontinuance of the drug, have been reported.

Other isolated reports of hypersensitivity reactions to 'FURADANTIN' include cases involving a fall in blood pressure, fever, asthmatic symptoms, muscular aches or jaundice. Occasionally a patient may show minor side-reactions such as headache or malaise. A small number of patients receiving 'FURADANTIN' therapy is reported to have developed peripheral neuritis. A pre-disposing condition in most of these patients was renal failure which often was accompanied by anaemia, diabetes, electrolyte imbalance, avitaminosis B or a debilitating disease. In the presence of these complications 'FURADANTIN' should be used only when indicated by in vitro sensitivity tests. If numbness or tingling occurs in any area, administration of the drug should be discontinued.

Reports of a few cases of haemolytic anaemia during 'FURADANTIN' therapy have been noted in the literature. A small percentage of patients have been found to have a deficiency of glucose-6-phosphate dehydrogenase. As a result, haemolysis may occur with certain drugs, such as primaquine and 'FURADANTIN'. Thus it is advisable to observe closely such patients receiving 'FURADANTIN' and to discontinue administration if there is any indication of this condition. It is reversible when the drugs is discontinued. If repeated or prolonged therapy is necessary, frequent chemical and cellular blood evaluations are strongly advised.

Allergy to nitrofurantion occasionally occurs and may manifest itself as a drug rash with or without eosinophilia, pyrexia or rigors. Rarely, a respiratory syndrome with bronchospasm and/or dyspnoea, cough and sometimes chest pain has been recorded. These symptoms have occasionally been associated with transitory pulmonary infiltration or pleural effusion.

Precautions

In the presence of impairment of renal function or acidosis, administer 'FURADANTIN' with caution as with any potent antibacterial agent. If employed under such circumstances the blood pH, CO_2 content or combining power, and urea nitrogen or nonprotein nitrogen should be followed closely.

'FURADANTIN' has been in widespread use now for several years. Clinical experience, and foetal studies in animals have shown no evidence to suggest that it may be a cause of congenital abnormality. However, it is recognised that caution should always be observed when prescribing for the pregnant patient, particularly during the first trimester.

Contra-indications

Anuria and oliguria are contra-indications to therapy with this compound because of inadequate excretion.

'FURADANTIN' should not be administered to infants under one month of age because of the possibility of producing a haemolytic anaemia due to immature enzyme systems (glutathione instability) in the early neonatal period.

Overdosage

Symptoms – Symptoms expected would be mainly extensions of side-effects. As 'FURADANTIN' is excreted rapidly in the urine administration of adequate amounts of fluid will hasten excretion of the absorbed drug.

Treatment – There is no specific treatment of overdosage. Treatment is essentially symptomatic – gastric lavage or induced vomiting.

Presentation

'FURADANTIN' 50 mg and 'FURADANTIN' 100 mg tablets are available in containers of 25, 100 and 250. Both presentations are allowable as Pharmaceutical Benefit Items, maximum quantity – 25 tablets, 1 repeat (restricted).

'FURADANTIN' Suspension containing 25 mg nitrofurantoin per 5 ml is available in bottles of 200 ml and to hospitals in bottles of 1,100 ml. FURADANTIN' Suspension is a Pharmaceutical Benefit Item, maximum quantity – 200 ml bottle, no repeats (restricted).

'FURADANTIN' Sodium is availalbe to hospital for intra ous use. Each sterile 20 ml vial contains sufficient crystalline 'FURADANTIN' Sodium to permit withdrawal of 180 mg 'FURADANTIN' as the sodium salt.

'Stelazine'

Description and Presentation

'STELAZINE' is available in the following forms:—

Dose Form	Strength Trifluoperazine (as the dihydrochloride)	Pack Sizes
Tablets	1 & 2 mg	50, 100 & 500
	5 mg	50, 100 & 500
	10 mg	100 & 500
'Spansule' Capsules	15 mg	50
Liquid	1 mg per 5 ml	100 ml
Liquid Forte	5 mg per 5 ml	100 ml & 1,100 ml
Ampoules	1 mg per ml	1ml & 2 ml in packs of 10
Suppositories	4 mg	5's

N.H.S. AVAILABILITY

'STELAZINE' 1 mg, 2 mg, 5 mg tablets, 'STELAZINE' liquid 1 mg/5 ml and 'STELAZINE' ampoules 1 mg/1 ml and 2 mg/2 ml are available as Pharmaceutical Benefits (Restricted).

Actions

'STELAZINE' is a phenothiazine tranquilliser. Compared to chlorpromazine, it has little effect on blood pressure or the action of other drugs; it has a slight adrenolytic activity. It is an antiemetic.

Indications

In low doses

To control excessive anxiety, tension and agitation as seen in neuroses or associated with somatic conditions. "STELAZINE' is also indicated in nausea and vomiting of various causes.

In high doses

For the management of manifestations of psychotic disorders, such as acute and chronic catatonic, hebephrenic and paranoid schizophrenia, psychoses due to organic brain damage, and toxic psychosis. 'STELAZINE' is also indicated for the control of the manifestations of manic depressive illness (manic phase) and of behaviour disorders in mental deficiency states.

Dosage

For Office Patients and Outpatients

Oral – Adults

1 or 2 mg twice daily. It is seldom necessary to exceed 6 mg a day except in patients with more severe conditions and in discharged mental patients.

Children

For children aged 3-5 years up to 1 mg as syrup a day according to body weight and physical condition. The dose for children of 6-12 years may be increased to a maximum of 4 mg a day. Dosage is based on a rate of 1 mg per 20 kg body weight.

Parenteral

For immediate control of symptoms or when oral administration presents difficulties, 'STELAZINE' may be given by deep intramu-

scular injection. Adults may be given 0.5 to 1 mg doses at intervals of 4 to 6 hours. Oral therapy should be substituted as soon as a satisfactory response has been achieved.

Rectal

The administration of a single 4 mg suppository usually provides symptomatic relief for 10-12 hours. A second suppository may be given several hours after the first but only in rare instances should dosage exceed 3 suppositories in any 24 hour period.

For Hospitalized Patients or Those Under Close Supervision

Oral – Adults

The usual starting dosage is 2 to 5 mg twice daily. If necessary dosage may be raised in increments of 5 mg daily at not less than 3 day intervals. When satisfactory control has been achieved, dosage may be reduced gradually until an effective maintenance level is established. Most patients will show optimum response on 15 or 20 mg daily although a few will require more.

Children

Dosage should be adjusted according to body weight and severity of symptoms. The usual starting dose for children aged 6-12 years is 1 mg twice daily. This may be increased gradually until symptoms are controlled or until side-effects become troublesome.

Parenteral

The usual adult dosage is 1 to 2 mg by deep intramuscular injection every 4 to 6 hours. More than 6 mg within 24 hours is rarely necessary. Only in exceptional cases should intramuscular dosage exceed 10 mg within 24 hours. In children 1 mg may be administered once or twice a day.

Notes on Dose Forms

'STELAZINE SPANSULE' capsules are equivalent mg for mg to 'STELAZINE' tablets but require only one dose every 24 hours. Thus a 15 mg 'Spansule' capsule daily provides the same result as a 5 mg tablet three times daily.

Given intramuscularly 1 mg of 'STELAZINE' is approximately equivalent to 3 mg orally.

One 4 mg suppository is approximately equivalent to 2 mg given orally. For Children uncooperative patients and those who have difficulty in taking tablets 'STELAZINE' liquids are often useful. They are pleasantly fruit flavoured and may be disguised in liquid or semi-solid foods. As 'STELAZINE' is an inherently long acting drug, oral doses may be conveniently given on a twice daily basis.

Contra-indications

'STELAZINE' should not be used in comatose or greatly depressed states due to central nervous system depressants, or in cases of existing blood dyscrasias, bone marrow depression and pre-existing liver damage.

Warnings and Precautions

Patients who have exhibited hypersensitivity reactions to any phenothiazine should not be treated with 'STELAZINE' unless the potential benefits outweigh the possible hazard.

'STELAZINE' may impair mental and/or physical abilities, especially during the first few days of therapy. Patients should be cautioned about activities requiring alertness, e.g. driving a car.

Care should be exercised when treating elderly or debilitated patients as some appear more prone to neurological side-effects.

Agranulocytosis, thrombocytopenia, pancytopenia and anaemia have been reported in patients receiving the drug. Jaundice of the cholestatic type of hepatitis of liver damage has been reported.

One result of therapy may be an increase in mental and physical activity. For example, a few patients with angina pectoris have complained of increased pain while taking the drug. Therefore, angina patients should be observed carefully and, if an unfavourable response is noted, the drug should be withdrawn.

In view of the possibility of hypotension large doses and parenteral administration should be avoided in patients with impaired cardiovascular systems, or those given spinal or regional anaesthesia. The drug should also be used cautiously in cases of circulatory collapse and the B.P. of patients undergoing surgery should be carefully monitored.

When treating prolonged or severe hypotension l-noradrenaline of phenylephrine should be used as vasoconstrictor agents. Phenothiazines may reverse the usual elevatory effect of adrenaline or other agents and may also produce hypotension in phaeochromocytoma patients.

Since certain other phenothiazines have been reported to produce retinopathy, lenticular and corneal lesions, the drug should be dis-

continued if ophthalmoscopic examination or visual field studies should demonstrate changes.

The antiemetic effect of 'STELAZINE' may mask signs of overdosage of toxic drugs or obscure the diagnosis of conditions such as intestinal obstruction and brain tumour. If agents such as sedatives, narcotics, anaesthetics, tranquillisers, or alcohol are used either simultaneously or successively with the drug, the possiblity of an undesirable additive depressant effect should be considered.

Because of the anticholinergic effects of phenothiazines they should be used with caution in patients with glaucoma or prostatic hypertrophy. With prolonged administration at high dosages, the possibility of cumulative effects, with sudden onset of severe central nervous system vasomotor symptoms should be kept in mind. It is therefore recommended that patients on long-term therapy at high doses may be evaluated periodically to decide whether the maintenance dose could be lowered or drug therapy discontinued.

Usage in Pregnancy

In pregnant patients any medication should be used only when the benefits outweigh the risks. If antiemetic or tranquilliser therapy is considered necessary under these circumstances then 'STELAZINE' is indicated.

Animal reproductive studies and clinical experience since 1958 have not demonstrated any teratogenic effect from 'STELAZINE'.

Adverse Reactions

At low dosage (2-4 mg daily) adverse reactions are infrequent, usually minor and transient, and unlikely to affect the course of therapy. Occasional instances of drowsiness, dizziness or stimulation may be observed Neuromuscular (extrapyramidal) reactions seldom occur in low-dose therapy.

Other reactions reported with phenothiazine compounds have included dry mouth, blurred vision, amenorrhoea, fatigue, muscular weakness, anorexia, skin reactions and lactation. Glycosuria, hyperglycaemai and blood dyscrasias have occasionally been seen.

Neuromuscular (Extrapyramidal) Reactions

These reactions are seen in a a significant number of patients receiving high dosages of 'STELAZINE' and other piperazine phenothiazine derivatives. Three groups of side-effects, all being recognized as extrapyramidal in origin may occur during the administration of these compounds. The incidence of such side-effects varies widely, but is lowest when the drug is increased gradulally to an optimal therapeutic level.

(1) Motor Restlessness (Akathisia)

Signs may include inability to remain still, tapping of feet, agitation and insomnia. Such motor restlessness may closely resemble the original neurotic or psychotic signs and it is important to recognise such side-effects for what they are and not to increase dosage until they have disappeared. These reactions may be controlled by a reduction of dosage and/or con-comitant administration of a barbiturate.

(2) Pseudo-Parkinsonism

Signs may include: mask-like facies; drooling; tremors; pillrolling motion; cogwheel rigidity; and shuffling gait. Reassurance and sedation are important. In most cases these symptoms are readily controlled when an anti-parkinsonism agent is administered concomitantly. (Note: Levodopa has not been found effective in pseudo-parkinsonism). Occasionally it is necessary to lower the dosage or to discontinue the drug.

(3) Dystonias

Signs may include: spasm of the neck muscles, sometimes progressing to torticollis; extensor rigidity of back muscles, extensor rigidity of back muscles, sometimes progressing to opisthotonos; carpopedal spasm, trismus, swallowing difficulty, oculogyric crisis and protruison of the tongue.

Despite their similarity to signs of such CNS diseases as tetanus, encephalitis or meningitis,particularly in children, these dystonias are readily reversible and need not cause undue alarm. They will usually subside within a few hours, and almost always within 24 to 48 hours, after the drug has been discontinued. Whilst they are usually related to high dosage they may occasionally occur as an idiosyncratic reaction in individuals given relatively low dosage of 'STELAZINE' for antiemesis or mild tranquillization. The signs tend to appear, fade and return again.

In mild cases, reassurance or a barbiturate is often sufficient. In more severe cases an anti-Parkinsonism agent (other than Levodopa) or intravenous caffeine and sodium benzoate 0.5 g

repeated if necessary or, injectable diphenhydramine usually produces rapid reversal of dystonic signs.

Persistent Tardive Dyskinesia

As with all antipsychotic agents, tardive dyskinesia may appear in some patients on long-term therapy or may appear after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterised by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g. protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tradive dyskinesia; anti-Parkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrom may not develop.

Overdosage

An accidental overdosage may cause Parkinson-like symptoms or symptoms of central nervious systems depression, agitation or restlessness. Treatment is symptomatic and supportive. Gastric lavage is helpful if performed early. The patient should be kept under observation and an open airway maintained, as involvement of the extrapyramidal mechanism may cause dysphagia and respiratory difficulty in severe cases. Parkinson-like symptoms may be alleviated by the administration of a suitable anti-Parkinsonism agent; antihistamines, such as diphenhydramine 25 mg intravenously, have also been used for this purpose. Painful dystonic muscular spasm may be relieved by the intravenous injection of caffeine and sodium benzoate 0.5 g repeated if necessary. The relief afforded by this measure usually occurs within 20 minutes. If hypotension occurs. the standard measures for managing circulatory shock (e.g. intravenous fluids and/or vasoconstrictors) should be initiated. If it is felt desirable to give a vasoconstrictor, noradrenaline or phenylephrine should be used. Other vasoconstrictors,

including adrenaline, should not be used as phenothiazine derivatives may reverse the sual elevating action of these agents and cause a further lowering of blood pressure.

Stimulants which may cause convulsions, such as picrotoxine or leptazol, should be aovided. Recommended stimulants are 'DEXEDRINE' and caffeine and sodium benzoate 0.5 g.

Roche Products Pty. Limited

Product Information

Valium Roche

Psychotherapeutic, muscle relaxant and anticonvulsant

Prescribing information

Composition

'Valium' Roche contains as active substance 7-chloro-1, 3-dihydro-1-methyl-5-phenyl-2H-1, 4-benzodiazepin-2-one (diazepam).

Properties

'Valium' Roche produces mental relaxation, a sedative hypnogenic effect and muscle relaxation.

Indications

Anxiety, tension, excitation, muscle spasm and motor unrest.

Contraindications

Patients with known hypersensitivity to the drug, acute narrow angle glaucoma, chronic oral use of the preparation in children under 6 months of age.

Precautions

Laboratory studies and extensive clinical experience have revealed no abnormal effects on foetal development due to 'Valium'. However, medication with 'Valium' Roche is not recommended during the first half of pregnancy. 'Valium' Roche may potentiate the central nervous depressant action of alcohol, sedatives, tranquillizers, antidepressants including MAO inhibitors, anticonvulsants, neuroleptics, analgesics, and anaesthetics.

Like all medicaments of this type, 'Valium' Roche may modify the patients reactions (driving ability, behaviour in traffic, etc.) to a varying

extent depending on dosage, administration and individual susceptibility. Cautions dosing must be employed when administering 'Valium' Roche to patients with organic cerebral changes (paritcularly arteriosclerosis) or with cardio-respiratory insufficiency. As a rule, parenteral administration should not be employed in these patients.

As with other agents which have anticonvulsant activity the possibility of provoking an increase in the frequency and/or severity of grand mal seizures may require an increase in the dosage of standard anticonvulsant medication. Abrupt withdrawal of 'Valium' Roche in such cases may be also associated with a temporary increase in the frequency and/or severity of seizures.

Because of isolated reports of reversible cholestatic jaundice, and transitory blood dyscrasias, periodic liver function tests and blood counts are advisable during long term therapy. 'Valium' Roche should only be given parenterally when adequate supervision of the patient is possible.

Adverse Effects

Adverse effects commonly reported were drowsiness, fatigue and ataxia. Infrequently encountered were confusion, depression, constipation, dysarthria, diplopia, headache, hypoactivity, hiccoughs, hypotension, incontinence, jaundice, nausea, changes in libido, changes in salivation, urinary retention, skin rash, synocpe, slurred speech, urticaria, tremor, vertigo, and blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, and stimulation have been reported and should any of these reactions occur the use of the drug should be discontinued. Isolated reports of neutropenia, and jaundice make periodic blood counts and liver function tests advisable during long term therapy. Minor EEG changes, usually low voltage fast activity, of no known significance have been reported.

Dosage

Average dosage for ambulatory patients: 2 mg three times daily by mouth.

Children:

6 months-3 years: ½-3 measures of syrup daily; 4-14 years: 2-6 measures of syrup daily; (1 measure = 2 mg)

Elderly and debilitated patients: 2 mg twice daily by mouth.

Hospital treatment of tension, excitation, motor unrest: 10-20 mg three times daily by mouth, continuted until acute symptoms subside.

Msucle spasm: 10-30 mg daily by mouth.

In obstetrical indications for continuation of therapy begun parenterally, see special packing slip for 'Valium' Roche ampoules.

These doses are only approximate and should be adapted to individual needs. For ambulatory patients engaged in their routine occupation a main evening dose of 5 mg and a smaller dose of 2 mg once or twice during the day is recommended

Presentation

White scored tablet 2 mg.
Yellow scored tablet 5 mg,
Light blue scored tablet 10 mg.
Syrup 2 mg per 5 ml,
Ampoules 10 mg per 2 ml.
Valium Roche = Trade Mark

Librium Roche

For the control of emotional functional and muscular disorders

Prescribing information

Composition

'Librium' contains as active substance 7-chloro-2-methylamino-5-phenyl-3H-1. 4-benzodiazepine 4-oxide.

Properties

In animals, the actions of 'Librium' include sedation, anticonvulsant effect, relaxation of striated muscle through inhibition of spinal reflexes, and stimulation of appetite. Clinically, 'Librium' exerts a prompt and marked influence on tesnsion and anxiety.

Indications

'Librium' is indicated whenever tension, fear and anxiety dominate the clinical picture or are the root of functional or somatic disorders. 'Librium' is also indicated in muscular spasm of psychogenic, neurogenic or myogenic origin.

Dosage and administration

Because of the wide range of clinical indications, the dosage of 'Librium' should be individually determined.

Average dose in adults: 20-40 mg daily.

Elderly or debilitated patients should receive 5-10 mg and only in exceptional cases more.

Children: 5-10 mg daily, increased if necessary to 20-30 mg, or in special cases more, daily.

Severe cases: 50-100 mg daily.
The tablets should be swalleowd whole with a little liquid.

Tolerance

In certain patients, particularly the elderly and debilitated, there have been occasional reports of ataxia and drowsiness: care is accordingly required in treating such persons.

With 'Librium' as with other psychoactive substances, patients should avoid taking alcohol while under the influence of the treatment, since the individual response cannot be foreseen. Like all medicaments of this type, 'Librium' may modify the patient's reactions (driving ability, behaviour in traffic, etc). to a varying extent depending on dosage, administartion and individual susceptibility.

Packings

Tablets 5 mg
Tablets 25 mg
Capsules 10 mg
Librium = Trade Mark

Mogadon Roche

Hypnotic

Prescribing information

Composition

The active substance of Mogadon is a benzodiazepine derivative: 1, 3-dihydro-7-nitro-5-phenyl-2H-1, 4-benzodiazepin-2-one (nitrazepam).

Properties

Taken in the evening Mogadon induces deep sleep lasting six to eight hours. Mogadon is well tolerated.

Indications

Nervous sleep disturbances due to irritability, overwork, conflicts,

anxiety, worry, tension and stress; organic sleep disturbances in conjunction with specific therapy.

Dosage

1-2 tablets (5-10 mg) before retiring. This average dosage may be increased if necessary up to 20 mg for inpatients.

Elderly patients			½-1 tablet
Children:	Infants	0-1 year	¼-½ tablet
	young children	1-6 years	½-1 tablet
	School children	6-14 years	1 tablet

The tablets may be swallowed whole, or dissolved in liquid.

Contraindications

Myasthenia gravis, in whicn the additional muscle relaxing effect of Mogadon could have deleterious consequences, severe chronic obstructive airway disease with incipient respiratory failure.

Precautions

With Mogadon as with other hypnotics, patients should avoid taking alcohol while under the influence of the treatment, since the individual response cannot be foreseen. Like all medicaments of this type, Mogadon may modify the patient's reactions (driving ability, behaviour in traffic, etc.) to a varying extent depending on dosage, administration and individual susceptibility. Since elderly patients are often particularly sensitivie to drugs, the dosage should be adapted accordingly. If Mogadon is combined with centrally acting drugs such as neuroleptics, tranquillizers, antidepressants, hypnotics, analgesics and anaesthetics, it should be borne in mind that their sedative effect may be intensified. This reinforcement can sometimes be made use of therapeutically. In elderly patients, especially with cerebrosclerosis or cardiorespiratory insufficency, caution is indicated, as with all hypnotics – and in rare cases paradoxical reactions in the form of restlessness and confusion may occur.

Reproduction studies

Laboratory studies and clinical experience with nitrazepam have revealed no signs of noxious effect on foetal development with dose administered in animals, corresponding to up to 500 times the normal therapeutic doses in humans. However, according to an established medical principle, nitrazepam should be given to women who are or who may become pregnant only when the potential benefits have

been weighted against possible hazard mother to and child. Radioactively labelled nitrazepam was administered to 5 lactating women to investigate the excretion of the drug in breast milk. Very low levels of the drug were detected – 0.05 to 0.1 ug of nitrazepam and/or metabolites per ml of milk – so that it seems unlikely that such small concentrations would produce any pharmacological or toxic effects.
Since these results deal with a rather limited number of patients and due to the impossibility of carrying out extensive clinical trials in this matter when administering Mogadon to nursing mothers, the potential benefits must be weighed against possible sedative effect in the child.

Side effects

Mogadon is well tolerated. In high doses (up to 200 mg orally) the muscular and psychosomatic relaxation, characteristic of the benzodiazepines is marked. Rare cases of ataxia have been observed. Respiration and blood pressure are unlikely to be affected by therapeutic doses of Mogadon.
Presentation
Scored tablets 5 mg
Mogadon = Trade Mark

Bactrim Roche

Broad-spectrum bactericide

Prescribing information

Composition

The active ingredients of Bactrim are : trimethoprim, (2, 4-diamino-5-[3, 4, 5-trimethoxybenzyl]-pyrimidine) and sulphamethoxazole (5-methyl-3-Sulphanilamido-isoxazole).

Each capsule or tablet contains:

Trimethoprim 80 mg
Sulphamethoxzaole 400 mg

5 ml of paediatric syrup contain:

Trimethoprim 40 mg
Sulphamethoxazole 200 mg

Properties

Bactrim is a bactericidal chemotherapeutic agent based upon recent scientific research. Its bactericidal action is the result of an original concept, namely, the sequential blockade of two enzymes acting within

the bacterial metabolic pathway of the biosynthesis of folinic acid. Bactrim is bactericidal at concentrations at which the components are usually separately bacteriostatic. It is frequently active against organisms which are resistant to one of the components, and further, the risk of bacterial resistance developing is reduced to a minimum. Bactrim is effective against a wide range of Gram-positive and Gram-negative organisms; for example, streptococci (including Group AB-haemolytic streptococci), pneumococci, staphylococci, Neisseriae, Salmonellae, Shigellae, *klebsiella/Enterobacter* group, *Vibrio cholerae* and *Bordetalla pertussis*. Bactrim pis particularly active against the problem organisms *Haemophilus influenzae, Escherichia coli* and *Proteus spp.*

Indications

Bacterial infections caused by a wide range of sensitive organisms.

Respiratory tract infections:

Acute and chronic bronchitis (including acute exacerbations of chronic disease), bronchiectasis, lobar and bronchopneumonia.

Renal and urinary tract infections:

Acute and chronic cystitis, pyelitis, pyelonephritis, urethritis.

Genital tract infections:

Male and female, including gonococcal urethritis.

Other infections:

Bactrim is indicated in septicaemias due to Gram-negative organisms as well as in other infections caused by a wide range of patholgenic bacteria, including typhoid fever.

Dosage

Adults and Children over 12 years old
Standard dosage: 2 capsules or tablets of Bactrim twice daily, morning evening after meals.

Minimum dosage: 1 capsule or tablet twice daily (see below).
Maximum dosage (for particularly severe infections): 3 capsules or tablets twice daily.

Children receive a dose corresponding to their ages.

PAEDIATRIC SYRUP.

Under 2 years — 2.5 ml twice daily.
2 to 5 years — 2.5-5.0 ml twice daily.
6 to 12 years — 5'0-10.0 ml twice daily.

The approximate dosage on a body weight basis is 4 mg/kg per day of trimethoprim and 20 mg/kg per day of sulphamethoxazole.

When used for the treatment of acute infections, Bactrim should be given for at least five days of until symptoms have subsided for two days. If Bactrim has to be administered for a period exceeding 14 days the minimum dosage is recommended (see above). In chronic chest infections, one tablet b.d. may be adequate for prophylaxis, but in some patients the standard dosage, two tablets b.d., may be necessary.

Adverse reactions

At the *recommended* dose, Bactrim is well tolerated, Nausea, vomiting, stomatitis and glossitis may occur. Spontaneous reversible haematological changes (mainly leucopenia and thrombocytopenia), occasionally marked, have been described. Aplastic anaemia and agranulocytosis have been reported.

Skin and systemic reactions may occur. Several cases of Stevens-Johnson Syndrome and toxic epidermal necrolysis (Lyell's Syndrome) have been reported.

Jaundice has also rarely occurred and has usually been mild and transient; frequently occurring in patients with a past hisotry of infectious hepatitis. The effect of Bactrim on human folate metabolism appears to be negligible.* One case with megalobalstic change in the bone marrow has been associated with long-term therapy on standard dosage.
On the whole, however, the nature of adverse reactions generally corresponds with what one would expect from a sulphonamide of moderately low toxicity.

Percentage incidence figures cannot be precise but have been estimated at 6.8% of cases treated in the published literature. * Sensitivity reactions and gastro-intestinal symptoms comprise nearly three quarters of the adverse reactions reported.*

Precautions

In cases with renal impairment a reduced or more widely spaced dosage is indicated to avoid accumulation of the drug. In such patients measurement of the plasma concentration of the drug is advisable. Regular blood counts are recommended wherever Bactrim is given for long periods. Patients should be examined monthly for haematological signs of folate deficiency. If such signs appear, Bactrim should be temporarily withdrawn. This is of particular importance in the aged, chronic alcoholics, patients with malabsorption syndrome, rheumatoid arthritis and those receiving anti-convulsants.

Contraindications

Bactrim is contra-indicated in patients showing marked liver parenchymal damage, blood dyscrasias or severe renal insufficiency, where repeated measurement of the plasma concentration cannot be performed, Bactrim should not be given to patients with a history of sulphonamide sensitivity.

The product should not be given during pregnancy nor should it be given to premature babies and the newborn during the first weeks of life. Babies should not be breast fed by women under Bactrim treatment.

Packings

Capsules
Tablets
Paediatric syrup
Bactrim = Trade Mark

*SALTER, A.J. Med. J. Aust. 1:2 (Special Supplement), 70 (1973).

Konakion - Roche

Vitamin K_1

Prescribing information

Properties

The presence of vitamin K (i.e. vitamin K_1 itself or substnaces with vitamin K activity) is essential for the formation within the body of prothrombin, factor VII and factor X. Lack of vitamin K leads to increased tendency to haemorrhage. When an antidote to an anticoagulant is necessary it is essential to use vitamin K_1 itself, as vitamin K analogues are much less effective.

Indications

Konakion is indicated in the treatment of haemorrhage or threatened haemorrhage associated with a low blood level of prothrombin or factor VII. The main indications are:

An antidote to anticoagulant drugs of the dicoumarol type

Prevention and treatment of neonatal haemorrhage

Dosage and Administration

Konakion may be taken orally or by injection.

For severe haemorrhae

The anticoagulant should be withdrawn and an intravenous injection of Konakion given *slowly* in a dose of 10 to 20 mg (1 or 2 ampoules). The prothrombin level should be estimated three hours later and, if the response has been inadequate, the dose should be repeated.

Less severe haemorrhage

Konakion is given orally in doses of 10 to 20 mg (1 to 2 tablets). The pleasant tasting tablets should be chewed thoroughly or allowed to dissolve slowly in the mouth. The prothrombin level is estimated 8 to 12 hours later and, if the response has been inadequate, the dose should be repeated. Intramuscular injections of Konakion may also be given in dose of 10 to 20 mg (1 or 2 ampoules), repeated if necessary.

Lowering of prothrombin to dangerous level but no haemorrhage

A dose of 5 to 10 mg Konakion may be given to bring the prothrombin level back to within safe limits. In such instances it is not usually necessary to discontinue the anticoagulant.

Note: Large doses of Konakion should be avoided if it is intended to continue with anticoagulant therapy. If haemorrhage is severe, a transfusion of fresh whole blood may be necessary whilst awaiting the effect of the vitamin K_1. Vitamin K_1 is not an antidote to heparin.

Treatment of new born infants

Prophylactic

1 mg by intramuscular injection.

Therapeutic

1 mg by intramuscular injection, repeated at 8-hourly intervals if necessary.

Precautions

Intravenous injections of Konakion must be administered *slowly* and reserved for potentially fatal haemorrhage due to overdosage of anti-coagulants of the coumarin and indanedione series. Not more than

40 mg should be given by intravenous injection during a period of 24 hours.

Konakion should be kept in a cool place protected from light.

Presentation

Konakion is the registered trade mark applied to pharmaceutical preparations containing vitamin K_1 phytomenadione, chemically described as 2-methyl-3-phytyl-1, 4-napthaquinone, available as follows:
Konakion ampoules 1 mg in 0.5 ml in boxes of 5.
Konakion ampoules 10 mg in 1 ml in boxes of 5.
Konakion tablets s.c. 10 mg in packings of 100.

COPPER "7"

Searle Laboratories,
Division of Searle Australia Pty. Ltd.,
8 West Street,
NORTH SYDNEY. 2060

The Time of Insertion

The Gravigard may be inserted easily at any time during the menstrual cycle. It is not necessary to delay insertion until the menstrual flow is in progress; however, the possibility of existing undermined pregnancy is less if insertion is made during or shortly following the menstrual period.

Preparing for Insertion

A routine bimanual examination should be carried out to determine size, position and mobility of the uterus.

NOTE: If the uterus is acutely anteverted, insertion may be found easier if the patient is in the left lateral position.

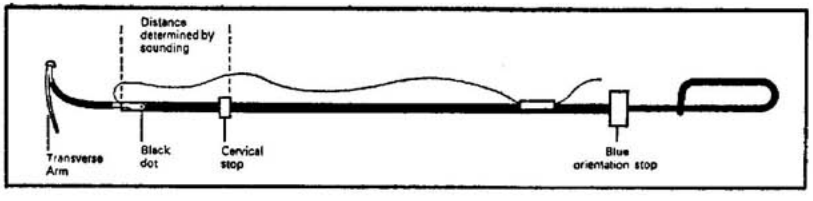
Sounding the Uterus. The cervix should be cleaned with an antiseptic solution and its anterior lip grasped with a tenaculum. Determination should be made of the depth and direction of the uterine cavity with a sound prior to insertion. After sounding the cervical stop should be adjusted so that its lower edge corresponds to the depth of the uterus as sounded. (See Step 1)

Loading the Gravigard. Sterile gloves should be worn and aseptic technique employed. Compress the two arms of the Gravigard by grasping them close to the mushroom cap and junction using sterile

forceps or while wearing sterile gloves and push fully into the introducer tube withdrawing the inserter rod a few centimeters to allow space for the device in the tube.

Caution should be exercised so as not to drop the Gravigard from the tube. The device should be loaded immediately prior to the insertion. Do not leave the device folded in the tube longer than a few minutes since the plastic may not maintain its proper shape.

Orient the Plane of the Device. Align the blue knob with the black dot on the Gravigard by grasping the inserter tube firmly while turning the blue knob. This procedure will insure that the device is positioned correctly in the uterus.



Step *1*: Insertion

Insertion is made by sliding the loaded tube with the black dot facing anteriorly or posteriorly through the cervical canal until the cervical stop reaches the cervix. Note: DO NOT FORCE! If resistance is felt before the fundus is reached, withdraw and reinsert. (Bending the tip of the metal rod may facilitiate insertion.)

Step *2*: Release Transverse Arm

The transverse arm of the copper bearing device is released by holding the handle in a stationary position and sliding the introducer tube backwards towards the operator not more than 25mm. (1 inch).

Step *3*: Position the Device at the Fundus

Hold the introducer tube steady, removing the thread retainer clip and push the insertor rod gently until the device reaches the fundus.

Step *4*: Trim Thread

The tube and rod are then removed and the excess protruding from the cervical canal is gently pulled into the vagina and cut approximately 3 cm from the cervix.

Indication

Intrauterine contraception.

Description

The plastic component of the Gravigard is composed of pharmaceutical grade polypropylene, homopolymer with barium sulphate added to render it radio-opaque. Its shape approximates the number 7, it is substantially smaller than previously available intrauterine devices.

Coiled around the vertical limb is a pure, virgin electrolytic copper wire. This wire provides a surface area of 200mm2. A polypropylene retrieval thread is fastened to the free end of the vertical limb of the Gravigard.

The Gravigard is supplied with a simple tubular polypropylene inserter. All components are sterile.

Contraindications

Pregnancy, abnormalities of the uterine cavity, history of repeated pelvic inflammatory disease, postpartum endometritis or infected abortion in the past three months, endometrial disease such as hyperplasia, polyps or suspected uterine malignancy.

Precautions

If pelvic infection occurs, which is unresponsive to treatment, consideration should be given to removal of the Gravigard. The Gravigard should not be inserted postpartum until after the first normal menstruation. The physician should weigh the benefits versus the risks of the Gravigard insertion in the immediate postabortion patient because of the relatively soft structure of the uterus. If pregnancy occurs with the Gravigard in situ, the risks of induced abortion through removal by the doctor should be considered. The possibility of ectopic pregnancy should not be excluded. Occasional perforations of the uterus (0.3%) have been reported, usually during insertion into patients who were less than two months post abortion or post partum. If penetration into abdominal cavity occurs, laparotomy should be performed and the device recovered. Local inflammatory reaction with abscess formation is a possibility if a device is left in the abdomen.

A report has appeared in the literature suggesting that a copper induced urticarial allergic skin reaction developed in a woman wearing a copper IUD. If symptoms of such an allergic response occur the patient

should be instructed to tell the consulting physician that a copper bearing device is being worn.

The use of microwave therapy in patients with metal prosthetics may cause heat injury to the surrounding tissue. Therefore, microwave therapy to the abdominal and sacral areas should not be used on patients wearing a Gravigard.

Side Effects

Rarely, postinsertion carmping, usually of no more than a few minutes' duration may occur. Transient spotting or bleeding or prolongation of menstrual flow may occur in the first few cycles. As with all IUD's post-insertion syncope may occur; however, due to the small diameter of the insertion tube, this is uncommon with Garvigard.

Copper in the Uterus

Copper concentration in the human endometrium normally range during the menstrual cycle from 8.6 to 24.3 micrograms per gram of protein. Minute increases of copper in the endometrium have been noted when the Gravigard is present; however, the blood levels of copper remained unchanged.

Current data do not demonstrate a significant effect of the Gravigard on endometrial histology. Theories on the mode of contraceptive action of copper at present centre on possible effects on enzymes participating in the process of implantation. The shape of the Gravigard and the insertion instrument and the directions for insertion were designed to facilitate insertion and to help reduce the possibility of complications at the time of insertion.

Replacement of Gravigard

Present information indicates that the efficacy is retained for at least 24 months. Until accurate data indicating a longer effective life become available, the Gravigard should be removed and a new one inserted on or before 24 months from the date of insertion.
Please read and follow the directions carefully.

SEARLE

Searle Laboratories,
Division of Searle Australia Pty Ltd.,
8 West Street,
NORTH SYDNEY. 2060

Metamucil and Instant mix Metamucil

Composition

psyllium hydrophilic mucilloid

Actions and Indications

Metamucil provides soft builk to restore natural bowel function and relieve constipation

Indications

Constipation, alone or associated with irritable bowal syndroms, mucous colitis, ulcerative colitis, peptic ulcer, diverticulitis, haemorrhoids, anorectal surgery, chronic illness, pregnancy and post parturition.

Also as an appetite-satisfying agent in obesity.

Contraindications

Intestinal obstruction
Fascal impaction

Dosage and Administration

(1) One rounded teaspoonful of powder or 1 sachet of Instant Mix in a glass of cool water, milk, fruit juice or other liquid.

(2) Stir briskly.

(3) Drink immediately. An additional glass of water is beneficial. Each dosage furnishes a negligable amount of sodium and about 14 calories.

Dosage: 1 to 3 times daily.

Presentation and Pack

Powder 100 gm
400 gm
Sachets (Instant Mix) 10's
100's

Floraquin

Indications:

Trichomonas vaginitis; mycotic vaginitis; non-specific and mixed vaginitis; leukorrhea and vaginal pruritus. Following cauterization

or conization of the cervix and in post-partum endocervicitis Floraquin may be used to provide an enviroment favorable to repair as well as a deodorant effect.

Dosage:

Two moistened tablets inserted high in the vagina before retiring daily for 2 or more menstrual cycles. Douches may be taken as indicated.

Contraindications:

Hypersensitivity to 8-hydroxyquinolines or iodine-containing preparations.

Warnings:

The use of any drug requires the physician to assess the benefits versus the risks for the patient.

Formula

Serenace, brand of haloperidol, is chemically described as 4'-fluoro-4-[4-hydroxy-4-(4-chlorophenyl) piperidino] butyrophenone.

Indications

Serenace affords rapid control of psychomotor agitation, anxiety, aggressiveness, excitement, hostility, hallucinations and delirium associated with acute and chronic psychoses.

Serenace is also a very potent anti-emetic.

Administration and Dosage

Serenace may be given intramuscularly, intravenously or by mouth, alone or in combination with anti-Parkinson drug therapy. Parenteral therapy is indicated for the urgent treatment of acute mania and acute psychotic agitation, particularly where a rapid effect is of important. Once adequate control of the patient is obtained, oral treatment can be given. For less urgent cases oral therapy may be prescribed from the beginning.

Since debilitated and geriatric patients may be more sensitive to Serenace the maximum and maintenance doses are generally lower for those patients than for nongeriatric adults. As Serenace is a long acting drug, the dosage may be administered once daily, although a number of authorities prefer a divided dosage, i.e. twice daily.

The following dosages are suggested as a guide:

Indications	Dosage
Agitation and Aggressiveness	For rapid control 5-10mg (1 to 2 ampoules) administered by i.v. or i.m. injection. This may be repeated 6-hourly until control is achieved when oral dosage may be substituted at 1.5-6mg daily. In senile agitation an oral dosage of 0.75-1.5 mg daily is usually sufficient.
Anxiety,	0.75-1.5 mg orally daily.
Schizophrenia acute and chronic	Initially 5-15mg i.v. or i.m. daily. Oral maintenance dose 1.5-6mg daily.
Mania and Hypomania	Rapidly controlled by an initial i.v. or i.m. injection of 5-20mg. Repeated 6-hourly as required. Oral maintenance dose 3-6mg daily.
Delirium Tremens	10-20mg i.v. or i.m. injection on admission followed by 5-10mg 6-hourly.
Anaesthesia	
(1) as a pre-medicant	3mg-5mg intramuscularly
(2) neuroleptanalgesia	5mg intramuscularly
Obstetrics	To allay anxiety during labour, the administration of 3mg intramuscularly is an average effective dose.
Anti-emetic	1.5-3mg oral or i.m.
Aggressive Behaviour in Mentally Retarded Children	1-3mg (10-30 drops) daily. Maintenance dosage for childrdn is usually 0.05mg per kilogram body weight daily, i.e. 1 Serenace drop morning and evening per 10lbs body weight.

Side Effects and Precautions

There are no known toxic effects associated with Serenace therapy. Where high dosage treatment is used, extrapyramidal side-effects (usually dystonic reactions or restlessness) may be encountered at an early stage. These can be controlled by reducing dosage or by the administration either of soporific or anti-Parkinson drugs, or pre-

vented by the concurrent use of anti-Parkinson drugs from the start of the treatment.

For immediate relief of dystonic reactions the parenteral route should be used. A pseudo-Parkinson rigidity syndrome may occur later during the course of treatment. This may be treated by giving anti-Parkinson agents. Serenace should be used with caution in patients with manifest lesions of the basal ganglia and in patients with arterioslcerosis who may have occult lesions of the basal ganglia. Where Serenace is given to patients with epilepsy it is important that the anti-convulsant therapy should be continued, as Serenace has no anti-convulsant activity.

Although Serenace has no soporific action, it should be appreciated that the drug will potentiate the soporific action of C.N.S. depressants. Pregnancy – the data available from animal and human studies do not suggest that haloperidol has any adverse effects on the foetus when taken during pregnancy. We do not, however, recommend the use of drugs in the first trimester since the possibility of some teratogenic effect occuring cannot be excluded. With Serenace as with other psycho-active drugs, patients should avoid taking alcohol since the individual response cannot be predicted.

Presentation

Oral

Tablets: Green, scored (6.9mm), uncoated tablets stamped "Searle" on one side, containing haloperidol 0.5mg in bottles of 50,100 and 500; white scored (7.2mm), uncoated tablets stamped "Searle" on one side, containing haloparidol, 1.5mg in bottles of 50, 100 and 1,000; also red, scored (8.0mm), uncoated tablets stamped "Searle" on one side, containing haloperidol 5.0mg in bottles of 100 and 500.

Liquid for oral use in 100 and 500 ml. bottles containing haloperidol 2mg per ml and in 15ml dropper bottles which deliver 20 drops per ml.

Parenteral

Ampoules: (amber coloured) of 1ml containing haloperidol, 5mg in boxes of 10 and 25.

General Pharmaceutical Benefits

*Ovulen 1/50**

Indications and dosage

Oral Contraception

One Ovulen 1/50 tablet daily for 21 days, starting on Day 5 of the menstrual cycle (where Day 1 is the first day of bleeding). The next course of tablets should be started after 7 clear days. Full instructions are given in each pack of 21 tablets.

Formula

The ingredients of Ovulen 1/50 are ethynodiol diacetate B.P. which is chemically described as 17α-ethinyl-4 oestrene-3β 17β-diol diacetate and ethinyloestradiol B.P.

Presentation

White, unscored, uncoated, pentagonal tablets stamped "Searle" on both sides, each containing ethynodiol diacetate 1mg and ethinylo-estradiol 50 mcg in wallets of 21 tablets.

Packs

One month's supply (21 tablets).

Notes

When Ovulen 1/50 is used for contraceptive purposes, it is not advisable to rely on this method along during the first week of the first course of tablets. Break-through bleeding with Ovulen 1/50 has only rarely besn reported after the third cycle. If it does occur the possibility of a cause unrelated to Ovulen 1/50 should be considered.

*Edulen 28**

Indications and dosage

Oral Contraception

Edulen 28 is a 28 day oral contraceptive plan which is taken continuously without a break bewteen cycles. The *first* tablet of the first course of Edulen 28 is taken from the WHITE SECTOR marked with the day corresponding to the *first* day of menstruation e.g. if bleeding commences on Tuesday this will be the patient's regular starting day.

She simply pushes out the Tuesday tablet (TUE) from the white sector of the pack and continues taking her tablets passing along the white sector into the gold sector following the directions of the arrows.

Tablets should be taken daily for 28 days when a new pack of Edulen 28 will be commenced the next day. The first tablet from the new pack

will always be taken from the white sector on the patient's regular starting day. Full instructions are enclosed in each pack.

Formula

The ingredients of the 21 active Edulen 28 tablets are ethynodiol diacetate B.P., which is chemically described as 17a-ethinyl-4 oestrene-3β 17β-diol diacetate and ethinyloestradiol B.P.

Presentation

21 white, unscored, uncoated, pentagonal tablets stamped "Searle" on both sides, each containing, ethynodiol diacetate 1 mg and ethinyloestradiol 50 mg plus 7 pink placebo tablets containing Dicalcium Phosphate 140 mg, which are larger in size than the active white tablets.

Pack

One month's supply (28 tablets).

Notes

When Edulen 28 is used for contraceptive purposes, it is not advisable to rely on this method alone during the first week of the first course of tablets. Break-through bleeding on Edulen 28 has only rarely been reported after the third cycle. If it does occur the possibility of a cause unrelated to Edulen 28 should be considered.

Other Searle Oral contraceptives available as general pharmaceutical benefits are

OVULEN 0.5, OVULEN 0.5/50, OVULEN 1,
OVULEN 2, CONOVID E.

The following information relates to all combined and sequential oral contraceptives:

1. *Contraindications*

   (i) Patients with thrombophlebitis, thromboembolic disorders, cerebrovascular accident, or with a past history of these conditions.

   (ii) Patients with liver disease or past history of cholestatic jaundice or pruritis of pregnancy.

   (iii) Patients with known or suspected carcinoma of breast or genital organs or suspected oestrogen-dependent neoplasia.

   (iv) Undiagnosed abnormal vaginal bleeding.

2. *Warnings*

   (i) The physician should be alert to the earliest manifestations of thrombotic disorders (thrombophlebitis, cerebrovascular disorders, pulmonary embolus and retinal thrombosis).
   Should any of these occur or be suspected the drug should be discontinued immediately.

   (ii) Discontinue medication pending examination if there is a sudden, partial or complete loss of vision, or if there is sudden onset of proptosis, diplopia or migraine. If examination reveals papilloedema or retinal vascular lesions, medication should be withdrawn.

   (iii) Since the safety of oral contraceptives in pregnancy has not been demonstrated, it is recommended that for any patient who has missed two consecutive periods, pregnancy should be ruled out before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule, the possibility of pregnancy should be considered at the time of the first missed period.

   (iv) Active ingredients of oral contraceptives have been detected in the milk of mothers receiving these drugs and the effect on breast fed infants has not been determined. Suppression of lactation may occur.

   (v) Oral contraceptive therapy should be discontinued at least six weeks prior to elective surgery because of the danger of thrombosis.

3. *Precautions*

   (i) Before prescribing oral contraceptives, physical examination is desirable including special reference to breast and pelvic examination.

   (ii) Under the influences of oestrogen-progestogen preparations, preexisting uterine fibroids may increase in size.

   (iii) Patients with conditions such as epilepsy, migraine, asthma and cardiac or renal dysfunction, require careful observation whilst on oral contraceptive therapy.

   (iv) Patients with a history of depression should be carefully observed and the drug discontinued if serious depression recurs.

(v) Because oestrogens may hasten epiphysial closure, oral contraceptives should be used judiciously in young patients in whom bone growth is not complete.

(vi) A decrease in glucose tolerance has been observed in a significant percentage of patients taking oral contraceptives.

(vii) A rise in blood pressure has been observed in some patients.

(viii) In break-through bleeding, when appearing for the first time in women who have been stabilised and previously well controlled and in all cases of irregular bleeding per vaginum, organic disease should be excluded.

(ix) Patients with disease affecting calcium and phosphorus metabolism should be carefully observed whilst on oral contraceptive therapy.

4. *Adverse Reactions*

In addition to those listed above, reactions which have been reported in women taking oral contraceptives include the following – Nausea, vomiting, Abdominal Cramps, Break-through Bleeding, Breast Changes (tenderness, enlargement and secretion), Changes in Menstrual Flow, Changes in Cervical Erosion and Cervical Secretions, Amenorrhoea During and After Treatment, Anovulation Post Treatment, Cholestatic Jaundice, Pruritis, Rash (allergic), Photosensitivity, Alopecia, Chlorasma, Erythema Multiforme, Erythema Nodosum, Haemorrhagic Eruption, Hirsutism, Headache, Migaine, Dizziness, Drowsiness, Changes in Libido, Changes in Appetite.

5. *Laboratory Data*

Physicians should be alerted to the fact that oral contraceptives may cause alterations in certain laboratory estimations.

(i) With the following tests, abnormal results may reflect an indirect interference with the test itself and not an impairment of organ function–

(a) Thyroid function – increase in PBI and butanol extractable protein bound iodine and decrease in T_3 values:

(b) Metyrapone Test – decrease in urinary 17-ketosteroids and 17-ketogenic steroids;

(c) Pregnanediol Determination – decrease in urinary pregnanediol levels.

(ii) With the following tests abnormal results may be significant–

(a) Liver function – increase in bromsulphalein (BSP) retention and serum transaminases (S.G.O.T., S.G.P.T.);

(b) Clotting factors – increase in factors VII, VIII, IX and X.

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